PHILOSOPHICAL TRANSACTIONS.

I. Croonian Lecture.—*Observations on Isolated Nerve (with particular reference to Carbon Dioxide).*

By Augustus D. Waller, *M.D., F.R.S.*

Received—Chapters I. and II., December 10, 1895; Chapter III., January 16, 1896; Chapter IV., May 28, 1896. Lecture delivered, March 12, 1896.

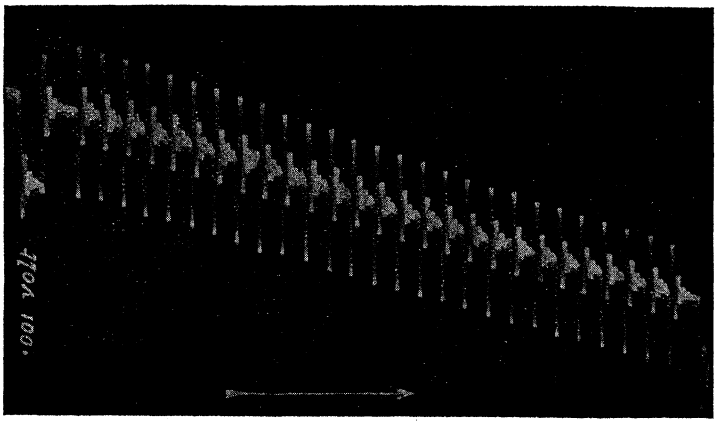
Contents.

Chapter I.—Introduction.

As was pointed out ten years ago by Wedenski, from telephone observations of the negative variation, by Waller, from galvanometric observations of the same, and by

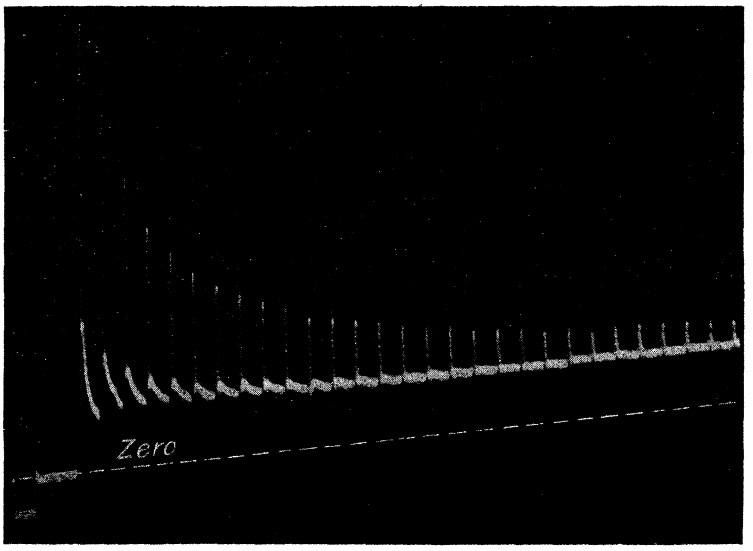
BOWDITCH from the observation of long-persisting excitability in the nerves of temporarily curarised mammals, nerve is now admitted to be practically inexhaustible. The apparent exhaustion of nerve when a nerve-muscle preparation is submitted to

Fig. 1. (Obs. 334.



Normal series of negative variations of current of injury of nerve. Excitation each minute for $\frac{1}{8}$ minute. The general decline south signifies a declining current of injury. Each negative deflection consists of a southward swing, followed by instrumental oscillations above and below the position of rest of the suspended magnets of a Thomson galvanometer. The deflection at the beginning of the series is by 0·001 volt.

Fig. 2. (Obs. 1501.)



Series of negative variations of current of injury of muscle, to show the contrast between their rapid decline and the undiminishing, or slightly diminishing, series of negative variations of nerve. Maximal indirect tetanisation for 7·5 seconds, at intervals of 1 minute. The deflection at commencement of record is by 0·001 volt. The rise of the base line is accidental. Dead-beat galvanometer.

prolonged excitation is, as was first demonstrated in BERNSTEIN'S experiment, not an exhaustion of nerve, but an exhaustion of the organ of intermediation between nerve

and muscle, which, as pointed out by WALLER, is the weak link in the neuro-muscular chain, being the point at which functional failure is first manifested, in fatigue, in nerve degeneration, in intoxication by curare (BERNARD), and at death.

Taking as the sign of action artificially excited in a nerve, the negative variation of its current of rest (DU BOIS-REYMOND) or current of injury (HERMANN), the long endurance by nerve of excitation excessive in strength and in length, is well shown by prolonged photographic records of the galvanometric indications, which exhibit little or no decline, in marked contrast with similar records of the negative variation of muscle which declines *pari passu* with the declining contraction due to fatigue of the motor end plates. This steady regularity of response renders nerve pre-eminently suitable to the systematic study of its experimental disturbance.

§ 1. *Method.*

The excised sciatic nerve of the frog is laid across two pairs of electrodes enclosed in a moist chamber through which gases can be passed; a tetanising current, of uniform duration and strength, excites the nerve once a minute through one pair of electrodes (of platinum, or unpolarisable); from the other pair of electrodes (unpolarisable) the nerve current and its negative variation at each minute are led off to a galvanometer.*

The tetanising current (lasting for $\frac{1}{8}$ minute) is effected by a revolving key in the primary circuit of a Berne standard induction coil; a reverser in circuit enables the direction of exciting currents to be reversed; these are "unmodified," *i.e.*, with the break shocks alone effective; two Leclanché cells (MUIRHEAD'S pattern) supply the primary coil; a short-circuiting key in this circuit is connected with a separate galvanometer, so that the current strength may be verified as constant; the secondary coil is fixed at a distance of 38 centims. from the primary, affording about 10 units of the Berne scale.

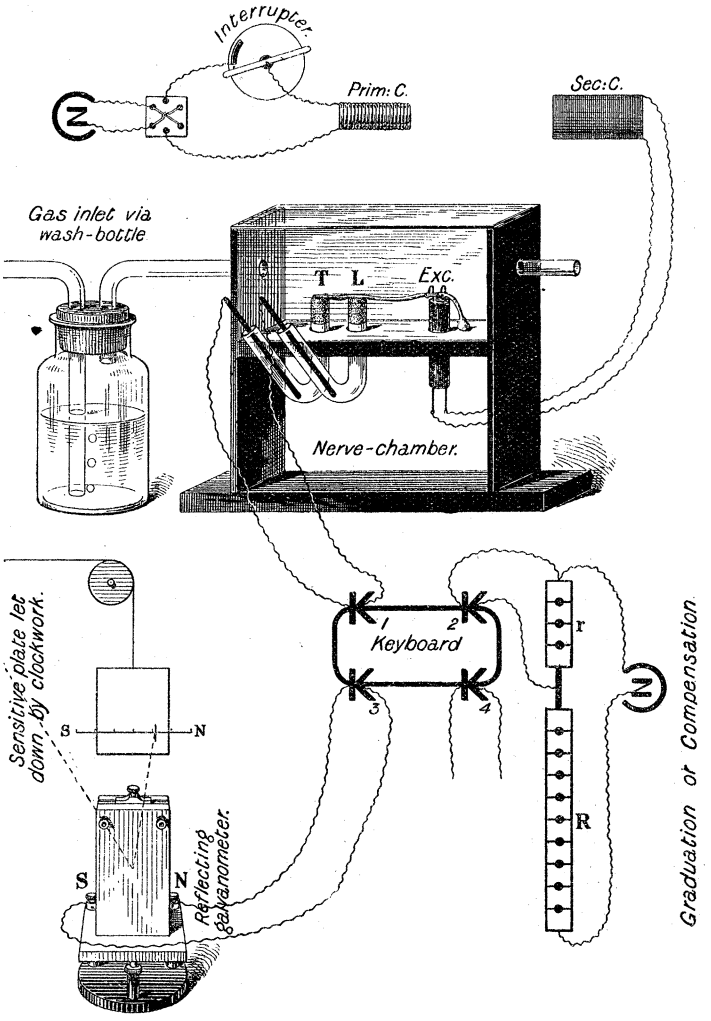
The nerve and galvanometer circuit, controlled by two short-circuiting keys (fig. 3, K_1 and K_3), connected with the nerve and galvanometer circuit respectively, could by a third key (K_2) be put in connection with a standard E.M.F. of $\frac{1}{1000}$ volt. A fourth key (K_4) is connected with a demonstrating galvanometer, the readings of which are observed on a screen in the usual way.

The moist or gas-chamber, with three sides of ebonite and three sides of glass, had a capacity of 30 cub. centims. The interval between the centres of the leading-off electrodes was 10 millims., that between the exciting electrodes was 3 millims., and that between the exciting and leading-off electrodes was 15 millims.

The galvanometer spot (image of a vertical slit in front of a paraffin lamp) fell upon a horizontal slit in a vertical box in which an "ordinary" photographic quarter plate was let down by clockwork.

* *Vide* note to Table D, p. 66. All the earlier records (*e.g.*, fig. 1) were taken with the partially-damped instrument; many of the later records (*e.g.*, figs. 66, 67, 68 and figs. 49 to 56) were taken with a dead-beat instrument.

Fig. 3.



Plan of Apparatus.

The *nerve-chamber* contains the nerve resting upon a pair of unpolarisable electrodes connected with K_1, and a pair of platinum electrodes, through which the nerve is excited. The wash-bottle serves to prevent drying of the nerve, and, in certain cases, is used to stop acid vapour.

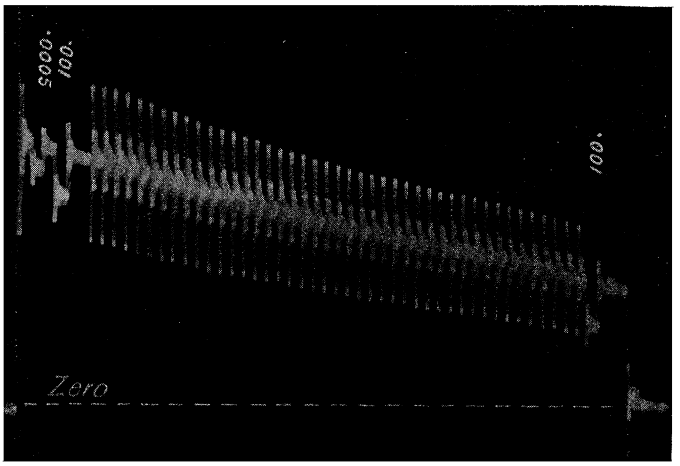
The *exciting apparatus* is represented above; the circular interrupter in the primary circuit revolves once a minute, and makes contact at a mercury pool for $\frac{1}{8}$ of each revolution. The vibrating interrupter of the coil starts as the circuit is completed, and the nerve is thus tetanised each minute for $7\frac{1}{2}$ seconds, at a frequency of 70–75 interruptions per second.

The *keyboard* is composed of four bridging keys: K_1 in connection with the nerve at T and L, K_2 with the compensator, K_3 with the recording galvanometer, and K_4 on occasion with a demonstrating galvanometer. When all the keys are closed, everything is short-circuited through them; the key-board is then simply a metallic ring. Opening K_1 and K_3 puts the nerve into connection with the galvanometer; opening K_2 lets into the nerve-circuit any suitable fraction of a volt from the cell and rheostats *r*.R. To take 0·001 volt from a Leclanché cell, *r* is taken = 10 ohms, and R = 14,690 ohms; to take a polarisation current of 0·01 volt, *r* is taken = 100 ohms, and R = 14,600 ohms, &c.

The connections in the diagram are represented such that the current of injury passes from left to right in the nerve, and in the galvanometer, viz., from T to L and from S to N, reading upwards in the final record. The compensation or graduation current reads downwards. A negative variation reads downwards. A positive variation reads upwards.

The order of procedure in experiment was as follows:—The nerve was dissected out, cut near the knee joint, and left in connection with a piece of spinal column to serve as a handle. It was then laid across the two pairs of electrodes and tested by a tetanising current, as above. Electrotonic effects were tested for by reversal of the exciting current, and by crushing the nerve in the interpolar region; unipolar effects by removing one wire; magnetic effects by removing both wires of the secondary coil; escape of primary current was also tested for and excluded by careful insulation. The excitatory deflection was, if necessary, reduced by shunting the galvanometer. The record was then started, a standard deflection by $\frac{1}{1000}$ volt being first taken through the nerve and galvanometer, shunted or not, to serve as a term of comparison, and as a witness to any possible alterations of resistance that might have occurred during experiment. This standard deflection was repeated whenever it seemed advisable by closure of the appropriate key. A sufficient

Fig. 4. (Obs. 309.)



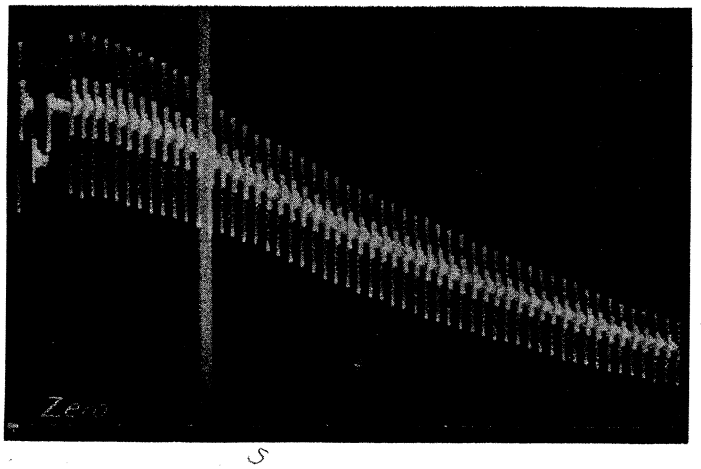
Similar series to that of fig. 1, on a more slowly moving recording surface. The zero line of the galvanometer is indicated; the standard deflections at beginning and end of the record are by $\frac{1}{1000}$ volt.

number of "normal" excitatory deflections having been recorded, usually 10 to 15, the experimental gas was passed through the gas-chamber, the time of passage of the gas being signalled by the light of a candle, about 10 feet distant, turned upon the slit behind which the sensitive plate was descending. In general the duration of an experiment was 1 hour, exceptionally $\frac{1}{2}$ hour, or 2 or 4 hours.

In earlier observations, while the first sciatic was under experiment, the second sciatic was left undisturbed in the animal and then prepared for the second experiment. In later observations both nerves were removed at the same time and kept in normal saline until required. (Compare figs. 5 and 6.)

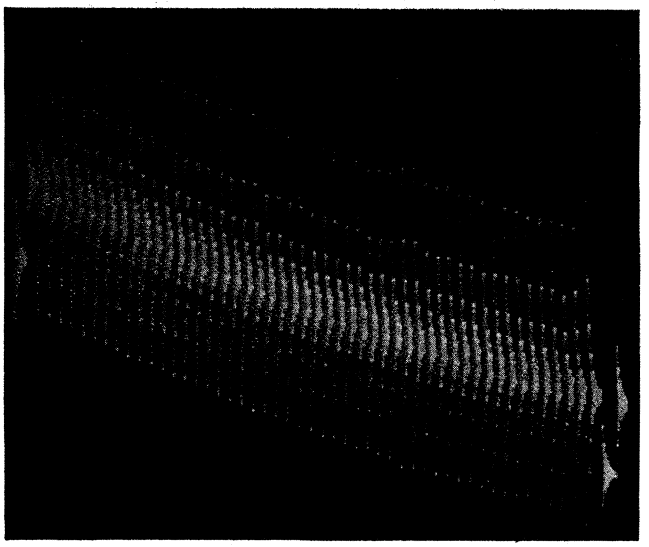
In order to secure the uniformity and easy legibility of record, the disposition of

Fig. 5. (Obs. 336.)



Similar series. Negative variation declining more rapidly than usual. Nerve left for about an hour in the pithed frog before use. The vertical white bar marks where nitrous oxide was passed into the nerve-chambers.

Fig. 6. (Obs. 708.)



Similar series. Negative variation of nearly constant magnitude. The excised nerve had been kept for 18 hours in physiological saline. The difference in the decline of the negative variation in this and in preceding figure is probably due to the action of CO_2 in the case of fig. 5, and the absence of such action in the case of fig. 6. The positive after-effect is well marked, giving an upward excursion of the magnet considerably in excess of the instrumental oscillation. (See pp. 66 and 84.)

connections adopted (transverse section of nerve to north terminal of galvanometer, longitudinal surface to south terminal) was such as to give:

NORTHWARD OR POSITIVE DEFLECTION BY THE NORMAL CURRENT OF INJURY.

SOUTHWARD OR NEGATIVE DEFLECTION BY THE NORMAL CURRENT OF ACTION.

And, for the same reason, the standard deflection by $\frac{1}{1000}$ volt was always taken in one direction, viz., southward.

The records read from right to left, north and south being their upper and lower borders respectively; positive deflections read upwards, negative deflections downwards.

With a fresh nerve, apparatus and connections disposed as above, the current of injury (north or above the zero line) gradually and steadily decreases (southwards or downwards), this decrease, according to ENGELMANN'S* observations and interpretation, is due to a diminishing negativity at and near the transverse section of the nerve; the negativity having fallen, may be raised again to or nearly to its original magnitude by a fresh transverse section; the original current and its decline are thus shown to be due to chemical action near the dying cut end rather than at the intact longitudinal surface. The line of decline during an hour's observation may be straight or concave to the abscissa, or convex at first, then concave to the abscissa.

The series of negative deflections (first swing south, followed in the case of the imperfectly damped galvanometer, by oscillations of the magnet north and south of the position of rest) frequently exhibits a progressive diminution, which one is at first thought inclined to regard as causally associated with the decline of the current of injury. Some such association may indeed obtain, but on a considerable number of observations is by no means clear or prominent, for with a large current of injury the negative deflection is sometimes small, and with a small current of injury it is sometimes large.

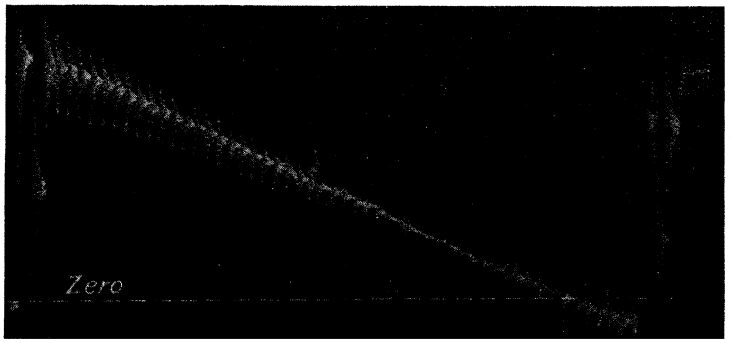
The progressive diminution of the current of injury might be significant of fatigue by the repeated excitations, or it might be due to a variety of accidental causes such as an unusually rapid death of a nerve, or a contamination of the clay electrodes by sulphate of zinc, or to acidity of the clay or of the salt solution. All these accidental causes excluded, it is easily seen that declining magnitude of the negative variation is not a fatigue effect, inasmuch as it proceeds uninfluenced by prolonged intermission of the excitation. Later experiments, as will be described below in detail, showed that one most effective cause of the decline was the previous action of carbon dioxide; and by yet other experiments it was rendered at least highly probable that another effective cause was the development of an opposite after-effect at and near the longitudinal lead-off.

In the earlier observations, the duration of which was, as a rule, limited to 1 or 2

* ENGELMANN. "Untersuchungen zur Lehre von der Muskel- und Nerven-elektricität," 'PFLÜGER'S Archiv,' vol. 15, 1877, p. 116.

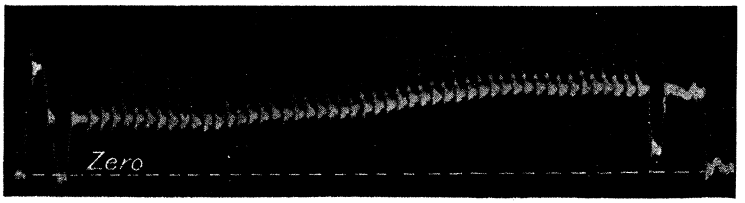
hours, an effect was noticed which was then regarded as somewhat exceptional (see figs. 7 and 8), but which in later observations, the duration of which has been often extended over several hours, has been recognised to be of frequent occurrence, viz., a reversal of the negative variation (south), and its replacement by a positive variation (north); the latter is shown to be a true physiological effect by the usual tests, by its susceptibility to anæsthetic vapours and to carbon dioxide; by the last-named

Fig. 7. (Obs. 279.)



Gradual reversal of the "normal" (south or negative) deflection, and appearance of a reversed (north or positive) deflection.

Fig. 8. (Obs. 842.)



Reversed or positive variation.

The nerve had been kept in physiological saline for $8\frac{1}{2}$ hours after removal, and had been used the hour previously for an experiment with CO_2.

When first observed the variation was composed of a small negative deflection followed by a large positive after-deflection. The variation in the figure is composed of a positive deflection (during excitation) followed by a second positive deflection (at the end of excitation).

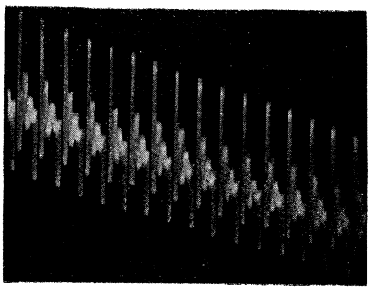
The period of the magnet is 15 seconds; the relation of its first swing from the position of rest to its first swing-back beyond that position is $\frac{+36}{-17}$. The duration of excitation is $7\frac{1}{2}$ seconds.

gas in particular such a positive variation is promptly and temporarily abolished. It was found not to be in any constant association with a reversed current of injury, which, indeed, was a quite exceptional phenomenon, due probably to some accidental injury at the longitudinal lead-off; the dying nerve at this point was then presumably negative to more nearly dead nerve on the side of the transverse lead-off.

A reversed variation, positive or northward in place of negative or southward, is difficult to explain, and any explanation is bound to be more or less hypothetical.

Several possibilities present themselves: (1) the reversal of excitatory deflection may be due to a reversal of action (or of greater action) in the parts of the nerve at and near the two leading-off electrodes; the normal south deflection depends upon action predominant at and near the longitudinal lead-off, the reversed north deflection might depend upon action becoming predominant at the transverse lead-off in consequence of some accidental and progressive injury near the longitudinal lead-off. Or the reversal might be (2) a phenomenon of the same order as HERING'S* "positive

Fig. 9. (Obs. 102.)



Portion of a series to show state of nerve intermediate between the first state (predominant south or negative excitatory deflection), and the third state (predominant north or positive excitatory deflection), viz., in which a small negative effect is followed by a larger positive after-effect.

The oscillation decrement in conjunction with the duration of excitation is such that a normal swing-back is equal to two-thirds of the original swing. In this case, therefore, a negative deflection of 7·5 has a positive oscillation of 5 millims., and the net positive after-effect is obtained by subtracting this value from the recorded after-effect, *i.e.*, by subtracting 5 from 12.

Nachschwankung." Or (3) the phenomenon and its reversal might be of an electrotonic nature. For reasons that will be given later, the second appears to me as the more probable explanation.

In the great majority of my experiments the negative deflection was normal, viz., a southward swing followed by a smaller northward swing-back in accordance with the oscillation decrement of the suspended magnet. But in a considerable minority of instances occurred what is, perhaps, hardly to be characterised as exceptional or abnormal, viz., the back-swing (north) was greater than the original (south) deflection (see fig. 9), and, in some cases, the original south deflection was small and sluggish and cut short by a sharp large deflection north. Instrumental fallacy was to the best of my knowledge excluded; moreover records occurred where, under the influence of carbonic acid and of ether, the relations between south and north variations were

* HERING, "Ueber positive Nachschwankung des Nervenstromes nach electrischer Reizung," 'Wiener Sitzungsberichte,' vol. 89, 1884, p. 219; HEAD, "Ueber die negativen und positiven Schwankungen des Nervenstromes," 'PFLÜGER'S Archiv,' vol. 40, 1887, p. 207.

gradually and temporarily changed in a way that could only be due to alterations developing in the nerve itself, and, finally, in some cases, most frequently in stale nerves and in drugged nerves (by urea, *e.g.*), the excitatory variation was throughout reversed (see fig. 8).

§ 2. *Concerning the Unfatiguability of Nerve. Relation between the Magnitude of Stimulation and the Magnitude of Electrical Effect.*

Two characteristics of the nerve-response should be mentioned at this stage, as being probably significant of a relative simplicity of this form of excitable matter, a simplicity that may be regarded as rendering nerve the most favourable among living tissues for the study of modifications by chemical reagents. These are :—

(1). The fact that in nerve the relation between magnitude of stimulus and magnitude of electrical effect is arithmetical within at least a considerable range, exceeding what, judged by the muscular response, may be termed the "physiological" range.*

(2). The fact just alluded to that the electrical effect increases with increasing magnitude of stimulus much beyond the physiological maximum effect as gauged by muscle.

Both these points have been reported upon elsewhere† and are alluded to here only inasmuch as they were at the outset of these experiments taken to be indicative of the relative simplicity of nerve-protoplasm, and having regard to the considerable range within which the electrical effects had been found capable of plus and minus variations (*e.g.*, between 2 and 20 or more stimulation units, as compared with a physiological range between 2 and 5 units), a stimulation strength of 10 units was uniformly made use of throughout these experiments.

Whether the adoption of this medium stimulation strength was essential or no, has not been fully determined; such preliminary experiments as were made, indicated weak stimuli as best adapted to the demonstration of increased electro-mobility, and strong stimuli to that of diminished electro-mobility. A medium strength was therefore adopted as a constant condition of experiment, at least throughout the first 800 observations; different strengths were used subsequently, and in some cases the stimulation strength for long series was selected three times the minimal value. Alterations of the direction of the deflection, with alterations of current strength, will be considered at a future stage.

No regard has been paid in these observations to a distinction between direct and

* This may be regarded as harmonising with the view that electrical excitation is due to electrolytic polarisation. I find, also, that HERMANN'S "polarisation increment" increases arithmetically with increasing strength of the polarising current.

† 'Proc. Physiol. Soc.,' 1895, p. 35. 'Brain,' 1895, p. 200. The second of the two points mentioned has previously been noticed by HERMANN ('PFLÜGER'S Archiv,' VII., 1873, p. 327).

indirect excitability, between the excitability at the point of stimulation, and the excitability aroused by propagation along the nerve ("conductivity").

Preliminary experiments were indeed made in order to test whether modifications

Fig. 10. (Obs. 101.)

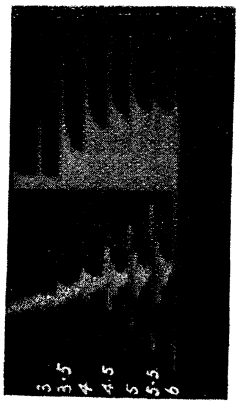


Fig. 11. (Obs. 134.)

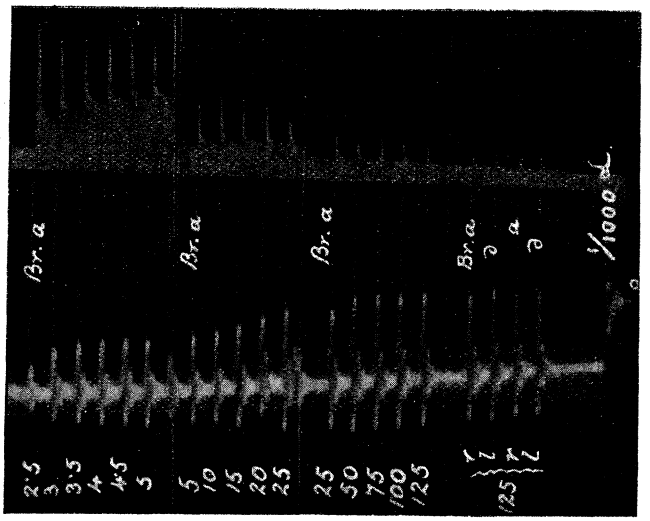


Fig. 10. Simultaneous record of the negative variation of nerve and of the contraction of muscle with increasing strength of stimulation (2·5 to 125 units of Berne coil).

The muscular response is dying off during the experiment, by lapse of time and by fatigue. The nerve response is kept up at a normal level, and increases in magnitude with increasing strength of stimulation (2·5 to 5 units of Berne coil).

The muscular response is dying off during the experiment, by lapse of time and by fatigue. The nerve response is kept up at a normal level, and increases in magnitude with increasing strength of stimulation, from 2·5 to 5, from 5 to 25, and more slowly up to about 50 units; at 125 units tests are made with reversal of direction of exciting currents, their inequality indicates that at this strength unipolar or electrotonic effects may become sensible; at lower strengths reversal of the exciting current was of no effect upon the magnitude of deflection; at the lowest, viz., minimal strengths differences were obtained in the nerve as well as in the muscular response signifying differences in excitation according to its direction (break currents).

N.B.—In figs. 10 and 11 the current of injury was downwards or south, and falls upwards or north; the negative variation was upwards.

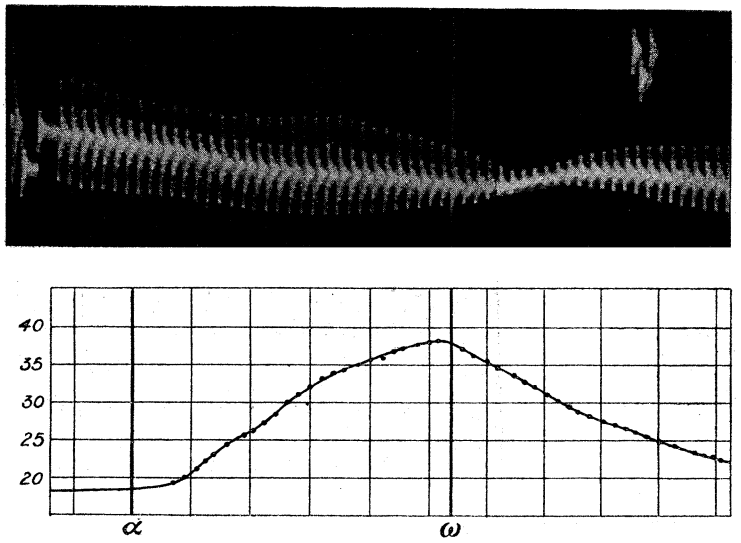
Fig. 11. Simultaneous record of the negative variation of nerve and of the contraction of muscle with increasing strength of stimulation (3 to 6 units of Berne coil).

The effect in nerve increases arithmetically with the stimulation, and continues to increase with increasing strength of stimulation beyond the point at which maximum tetanus of muscle is produced. The maximum contraction grows less in the progress of experiment.

by reagents were at the leading-in (stimulating) electrodes, or at the leading-out (galvanometer) electrodes. These experiments will be described in connection with anæsthetics. At present, it will be sufficient to state that the modifications of excitability (or of "electro-mobility"), were modifications principally at the leading-

out electrodes, by which the entire nerve was, so to say, "sampled." Other preliminary experiments were made (see fig. 23) to determine whether such propagated effects behaved in any way differently from directly excited effects; the two circuits (exciting and galvanometer) were conjoined so that the nerve, secondary

Fig. 12. (Obs. 773.)



Effect of a rise of temperature from 18° to 38° upon the nerve response. The delay between the maximum temperature reading and the maximum diminution of the nerve response is probably owing to the "lag" of the apparatus between the nerve and the source of heat. The nerve was in a glass chamber in a copper oven, under which a small spirit-lamp flame was placed at α and removed at ω. The temperature chart below is that of corresponding readings each minute of a thermometer in the oven. No marked effect occurs with a rise of at least 10°; with a rise to 38°—viz., of 20°—the response diminishes; with the subsequent fall of temperature the response increases again.

The record is given in illustration of the fact that the response of fresh nerve is not affected by slight variations of temperature. The effects of considerable variations, as regards the character and sign of the response, will be described and considered in connection with the detailed study of positive and negative effects of excitation.

N.B.—The actual magnitudes of excursions, *e.g.*, in fig. 13, as compared with fig. 12, are of no account as indicative of magnitudes of nerve-effects in terms of potential. The resistance differs in different nerves, and the galvanometer is more or less shunted. The magnitudes of excursions must in all cases be compared with the magnitude of the standard excursion by 0·001 volt taken at beginning and end of each observation.

coil, and galvanometer formed one circuit, with the unpolarisable electrodes serving as leading-in and leading-out electrodes. The nerve was then excited with alternating currents (make and break) weak enough not to disturb the galvanometer, yet strong enough to disturb the nerve (viz., five stimulation units), first in one, then in the opposite direction, and the negative variation by direct excitation was

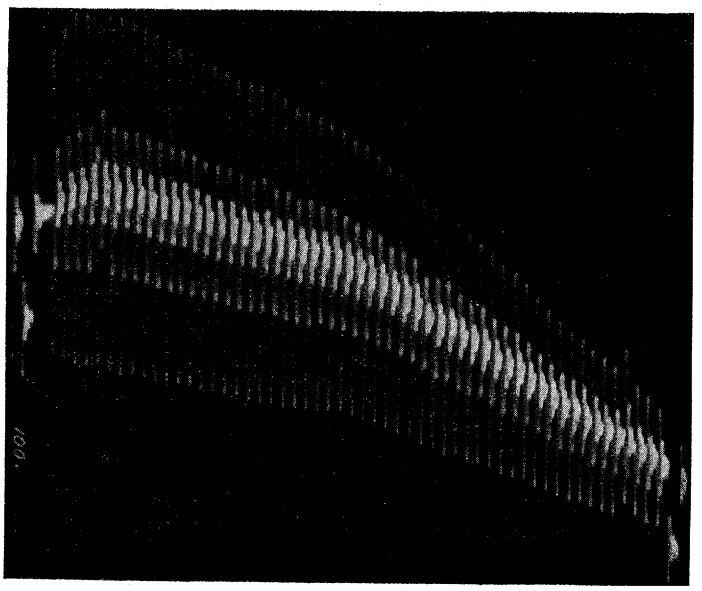
compared and found to be identical with the negative variation by propagated excitation. This conjoint method might, indeed, have been systematically followed, but for several reasons it was preferred to apply stimulation to a separate part of the nerve, regarding the latter simply as a strand of excitable protoplasm to be disturbed at one point, the disturbance being measured at another point, under a variety of chemical conditions.

§ 3. *Temperature, Moisture, and Atmospheric Pressure.*

Preliminary observations were made as to the influence of temperature and moisture—of the former by enclosing the gas-chamber in an oven of which the temperature was gradually raised and lowered, of the latter by leaving the gas-chamber open with the nerve and electrodes exposed to the air.

As regards temperature, it was found that between 30° and 40° the excitability

Fig. 13. (Obs. 825.)



Effect of drying by evaporation upon the response of nerve. Stimulation as usual, 10 units tetanisation for $\frac{1}{8}$ minute at each minute.

Nerve kept in physiological saline for $2\frac{1}{2}$ hours after excision; lid of gas-chamber removed. In the absence of evaporation such a nerve would probably have given an uniform series, as in fig. 6. The relative magnitudes of the deflections by 0·001 volt at beginning and end of the series indicate to what extent the diminishing deflection is due to increasing resistance of the nerve.

declined and disappeared, to return, however, with subsequent fall of temperature (fig. 12).

As regards moisture, it was found that the excitability of a drying nerve progressively diminishes (fig. 13), being restored, however, by subsequent moisture.

The possible influence of alterations of barometric pressure was also examined on nerves in nerve-chambers, within an iron vessel, in which the pressure was raised to 3 atmospheres and depressed to $\frac{1}{3}$ atmosphere. The results of these alterations were throughout negative.

It was therefore concluded that small changes of temperature, of moisture and of pressure, such as may occur in the gas-chamber, are negligible.

Papers referred to.

WEDENSKI. "Die telephonischen Wirkungen des erregten Nerven." 'Cbt. f. d. medic. Wissenschaft.,' 1883, p. 465.

WALLER. "Experiments and Observations relating to the Process of Fatigue and Recovery." 'British Medical Journal,' 1885 and 1886.

BOWDITCH. "Note on the Nature of Nerve Force." 'Jl. of Physiology,' 1885, p. 133.

„ "Ueber den Nachweis der Unermüdlichkeit des Säugethiernerven." 'DU BOIS-REYMOND'S Archiv,' 1890, p. 505.

BERNSTEIN. "Ueber die Ermüdung und Erholung der Nerven." 'PFLÜGER'S Archiv,' vol. 15, 1877, p. 289.

ENGELMANN. "Vergleichende Untersuchungen zur Lehre von der Muskel- und Nervenelektricität." 'PFLÜGER'S Archiv,' vol. 15, 1877, p. 116.

HERING. "Ueber positive Nachschwankung des Nervenstromes nach electrischer Reizung." 'Wiener Sitzungsberichte,' vol. 89, 1884, p. 219.

HEAD. "Ueber die negativen und positiven Schwankungen des Nervenstromes." 'PFLÜGER'S Archiv,' vol. 40, 1887, p. 207.

WALLER. "Points relating to the Weber-Fechner Law; Retina, Nerve, Muscle." 'Brain,' 1895, p. 200.

„ "The Quantitative Relation between Stimulation and Negative Variation of Nerve." 'Proc. Physiol. Soc.,' June, 1895.

HERMANN. "Untersuchung über das Gesetz der Erregungsleitung im polarisirten Nerven." 'PFLÜGER'S Archiv,' vol. 7, 1873.

Chapter II.—The Influence of Carbon Dioxide.

§ 1. *Action of "much" CO_2 and of "little" CO_2 on Normal Nerve.*

Reagents in the state of gas or vapour are obviously the most convenient means of testing chemical modification of nerve according to the method described in the previous chapter, inasmuch as the nerve remains undisturbed throughout observation.

Chloroform, ether, and other anæsthetics were first tested, with results such as might be anticipated from their known action on man and animals. These were demonstrated at the November meeting of the Physiological Society, and will be fully reported upon in a future communication.

Carbon dioxide, carbon monoxide, nitrous oxide, oxygen, methane, coal gas, hydrogen, hydrogen sulphide, chlorine, ammonia, hydrocyanic acid, amyl nitrite (impure) were next tested, with results partly expected, partly unexpected. Thus, *e.g.*, coal gas, oxygen, carbon monoxide, and nitrous oxide produced no effect, while more or less pronounced effects were obtained from all the other gases named.

Among these various gases carbon dioxide afforded results of most physiological interest, as, indeed, might have been expected in the case of one of the chief terminal products of protoplasmic action.

Detailed study of the action of CO_2, in varying amounts and under various conditions, afford results of great simplicity and experimental certainty, serving as a foundation upon which rest: 1. Experimental evidence of a production of CO_2 by active nerve. 2. Experimental separation of two opposed movements (of assimilation and dissimilation, or of polarisation and depolarisation ?) in nerve, oppositely influenced under certain conditions: (a) by CO_2 from outside, (b) by CO_2 produced within nerve itself.

These two subjects cannot, however, be discussed with any advantage until the action of CO_2 upon isolated nerve has been fully ascertained and considered; this is the more necessary in that its effects are not at all what might have been anticipated from the frequently quoted experiment of Grünhagen* concerning the action of CO_2 upon the nervous conductivity.

The characteristic action of CO_2 upon normal (or nearly normal) isolated nerve will be best and most briefly presented by the following summary notes of fifty consecutive† galvanographic records, together with a few selected representative records, and followed by the conclusions to be drawn from these fifty observations.

* Grünhagen, "Versuche über intermittirende Nervenreizung," 'Pflüger's Archiv,' vol. 6, 1872, p. 180.

† Szpilman and Luchsinger ('Pflüger's Archiv,' vol. 24, 1881), extended Grünhagen's experiment and contradict his conclusion.

TABLE A.—Summary of Observations on the Action of Carbon Dioxide upon Normal Nerve.

No.	Plate No.	Duration of CO_2 current.	Negative variation.	Remarks.
1	172	12 minutes	*Before:* 15 millims. . . . *During:* augmentation to 20, giving way to diminution to 5 *After:* augmentation to 70, gradually declining	$\frac{1}{1000}$ volt gave a deflection of 25 millims.
2	173	2 ,,	*Before:* 15 millims. . . . *During:* 25 ,, *After:* 50 ,,	Same nerve.
3	182	8 ,,	*During:* abolition. . . . *After:* augmentation	Base-line rises north.
4		4 ,,	*During:* abolition. . . . *After:* augmentation	25 minutes' interval between first and second trials.
5	183	3 ,,	Slight after-augmentation .	On a previously fully etherised nerve.
6	190	3 ,,	Ditto	On a previously chloroformed nerve.
7	194	4 ,,	*Before:* 20 millims. . . . *During:* 0 ,, *After:* 60 ,,	Base-line rises north after passage of CO_2.
8	195	3 ,,	*During:* abolition. . . . *After:* augmentation	7 minutes between the two trials.
		3 ,,	Ditto	
9	196	2 ,,	Ditto	Same nerve.
10	198	2 ,,	Ditto	Base-line falls during, rises after.
11	200 (fig. 14)	2 ,,	Ditto	Typical effect, viz., primary abolition, secondary augmentation.
12	240	2 ,,	No effect	Nerve of a naturally dead frog.
13	245	1 ,,	Expired air: augmentation	Presumably a CO_2 effect.
		1 ,,	Room air: no effect	
		1 ,,	Expired air: augmentation	
		1 ,,	Room air: no effect	
14	413	3 ,,	*Before:* 4 millims. . . . *During:* 0 ,, *After:* 30 ,,	30 minutes after CO_2 the original S. deflection gave way to N.
15	416	1 ,,	Similar to No. 1	After $CaBr_2$.
16	432	1 ,,	Typical effect	After $\begin{cases} CHCl_3. \\ CH_2Cl. \end{cases}$
17	437	1 ,,	Ditto	Ditto.
18	464	1 ,,	Diminution followed by augmentation	After recovery from ether depression.
19	521	1 ,,	Ditto	
		1 ,,	Ditto	
20	522	10 ,,	Augmentation for 2 minutes, giving way to abolition for 10 minutes, followed by augmentation *Before:* 15 millims *During:* 20 . . . . 0 *After:* 60 . . . . declining	
21	537 (fig. 15)	5 ,,	Primary augmentation, diminishing, followed by secondary greater augmentation	The negative S. effect is relatively larger, the positive N. after-effect is relatively smaller after the passage of CO_2.

TABLE A.—Summary of Observations on the Action of Carbon Dioxide upon Normal Nerve—(continued).

No.	Plate No.	Duration of CO_2 current.	Negative variation.	Remarks.
22	561	2 minutes	Primary diminution, secondary augmentation	After recovery from ether. The normal S. gradually gave way to an N.S. deflection.
23	574	1 ,,	No effect	After ammoniated chloroform. No recovery. Twice tested.
24	579	1 ,,	Typical effect	
25	598 (fig. 20)	Expired air 2 minutes	Primary augmentation . .	To test the effect of expired air; 3 per cent. CO_2.
26	599	1 ,,	Abolition followed by slow recovery. No augmentation within 30 minutes	The original deflection was N.; after CO_2 it became N.S.N., gradually giving place to N. The plate was twice used, and cannot therefore be precisely read. A variation of similar character N.S.N. is recorded on Plate 758.
27	607	1 ,,	Typical effect	
28	610	1 ,,	Ditto	
29	627	13 ,,	Ditto	
30	633 (fig. 17)	$2\frac{1}{2}$,,	Ditto	
31	638	2 ,,	Ditto	
32	641	$2\frac{1}{2}$,,	Ditto	
33	646	1 ,,	Abolition followed by recovery; no subsequent augmentation	Nerve of a frog killed some hours previously; left *in situ*, *i.e.*, subject to the reducing action of the tissues.
34	647	3 ,,	Primary augmentation followed by secondary greater augmentation	CO_2 in very slow stream. At about the 5th minute after CO_2, it was noticed that the S. effect was augmented while the N. after-effect was diminished; later both were increased.
35	648	3 ,,	Primary diminution, secondary augmentation	After imperfect recovery from ether.
36	650		Slight primary augmentation, giving way to diminution, followed by secondary augmentation	Double gas-chamber. CO_2 passed through upper or excitation compartment. Greater augmentation of S. than of N.
37	651	3 ,,	Primary augmentation, giving way to diminution, followed by secondary augmentation	Double gas-chamber same nerve. CO_2 through lower or lead-off compartment. Effects similar to but more pronounced than those of previous experiment.
38	652 (fig. 18)	1 ,,	Primary augmentation, diminishing slightly, followed by secondary augmentation	Double gas-chamber. CO_2 through lead-off half. Augmented S. with diminished N.
39	653	1 ,,	Slight diminution, followed by slight augmentation	Double gas-chamber. Same nerve. CO_2 through leading-out half.
40	654	100 cub. centims.	Slight augmentation . . .	Base-line rises.
		100 ,, ,,	Ditto	Ditto.

TABLE A.—Summary of Observations on the Action of Carbon Dioxide upon Normal Nerve—(continued).

No.	Plate No.	Duration of CO_2 current.	Negative variation.	Remarks.
41	671 (fig. 21)	. . . . .	. . . . .	To test a presumable CO_2 effect in a nerve left subject to the action of surrounding tissues. Frog killed 18 hours previously. The negative deflection is very large (= about 0·004 volt) as compared with the usual normal (= about 0·001 volt) and declines very rapidly.
42	672 (fig. 22)	. . . . .	Diminution followed by imperfect recovery	Second nerve of same frog. Imperfect CO_2 effect upon the presumably already carbonised nerve.
43	673	1 minute	Ditto	Same nerve.
44	674 (fig. 19)	1 ,,	Slight primary augmentation, declining, followed by large secondary augmentation	A perfectly fresh nerve, original deflection = 0·0013, after CO^2 = 0·0035, the S effect is greater, while the N after-effect is still smaller.
45	740	4 ,,	Abolition followed by small augmentation	Frog killed 2 hours, nerve *in situ*. The same nerve after a normal saline bath of 1 hour, subsequently gave a marked CO_2 effect by 5 minutes tetanisation.
46	747	2 ,,	Primary augmentation . .	Greater augmentation of S.
		2 ,,	Primary diminution followed by secondary augmentation	Greater diminution of N.
47	752	1 ,,	Abolition followed by augmentation	Frog killed 6 hours, nerve in saline. Unequal and asynchronous effects on S. and N.
48	786	1 ,,	Primary augmentation	
50	869 (fig. 16)	2 ,,	Augmentation	Large fresh nerve; very slow stream of CO_2.

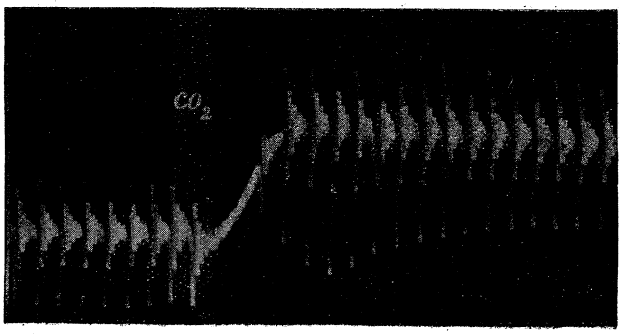
The conclusions to be drawn from these data (which were supplemented by many others unrecorded) are as follows :—

(1). *The typical effect of carbon dioxide upon normal nerve is a brief abolition followed by a prolonged augmentation of excitability* (figs. 14 and 17).

This result may nearly always be demonstrated, using fresh nerves of a recently killed healthy frog, and running CO_2 in full stream through the gas-chamber during 1 or 2 minutes.

With stale nerve, left in the animal killed 2 or 3 hours, or longer, 12 to 18 hours, the primary effect of CO_2 (diminution) is more marked and prolonged, the secondary effect (augmentation) is less marked (fig. 22). The negative deflection is large and rapidly declines.

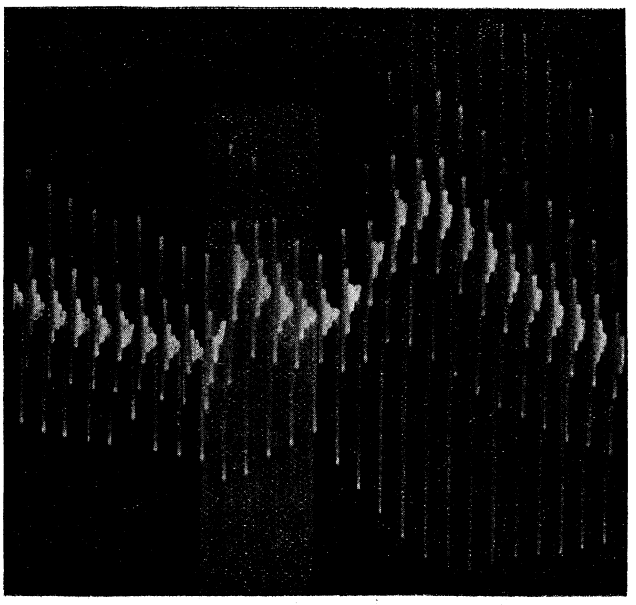
Fig. 14. (Obs. 200.)



Effect of CO_2 in full stream on normal freshly excised nerve. "Typical effect," viz., primary abolition followed by secondary augmentation of excitability.

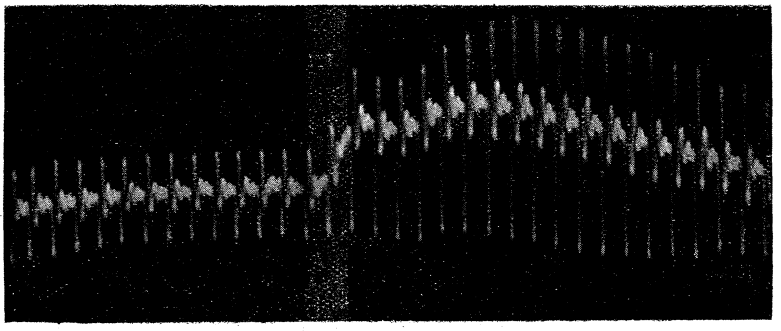
The gas-chamber containing the nerve measures 30 cub. centims. The nerve is tetanised each minute for $7\frac{1}{2}$ seconds by ordinary make and break shocks at a frequency of about 70 per second, and a strength of 10 units of a Berne coil, supplied by two Leclanché cells. Interrupter in primary circuit. Time of passage of CO_2 through the nerve chamber indicated by the vertical bar across the record. Deflection before CO_2 = that of 0·0006 volt; after $CO_2 = 0$; 6 minutes later = 0·0012 volt. The "lag" of the CO_2 effect is probably due to diffusion time of the gas in contact with the nerve.

Fig. 15. (Obs. 537.)



Effect of a "moderate" quantity of CO_2 on nerve under similar conditions. Primary augmentation, diminishing; secondary further augmentation.

Fig. 16. (Obs. 869.)



Effect of a "small" quantity of CO_2 on nerve under similar conditions. Primary augmentation, in this instance, checked by a very slight diminution. Compare with figs. 5 and 6.

Fig. 17. (Obs. 633.)

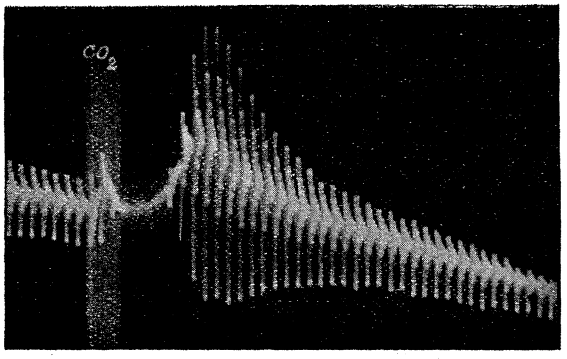


Fig. 18. (Obs. 652.)

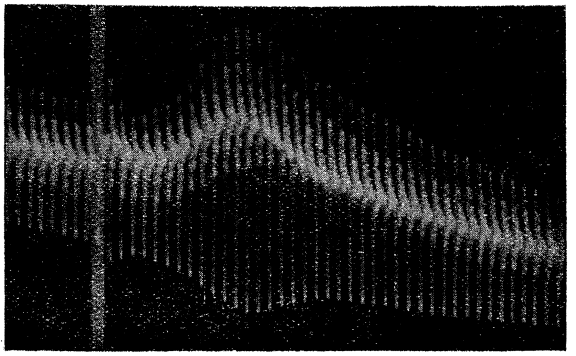
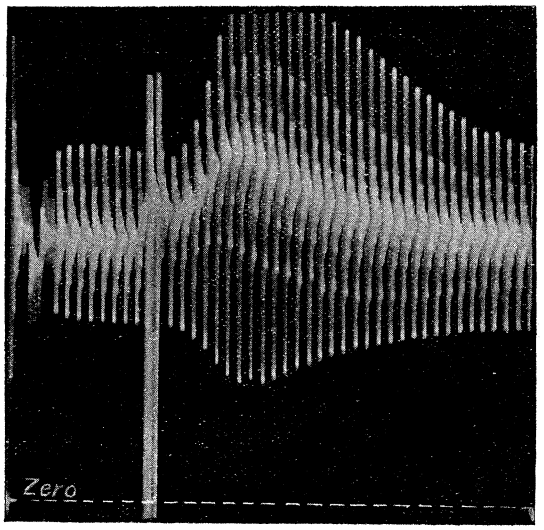


Fig. 19. (Ob. 674.)



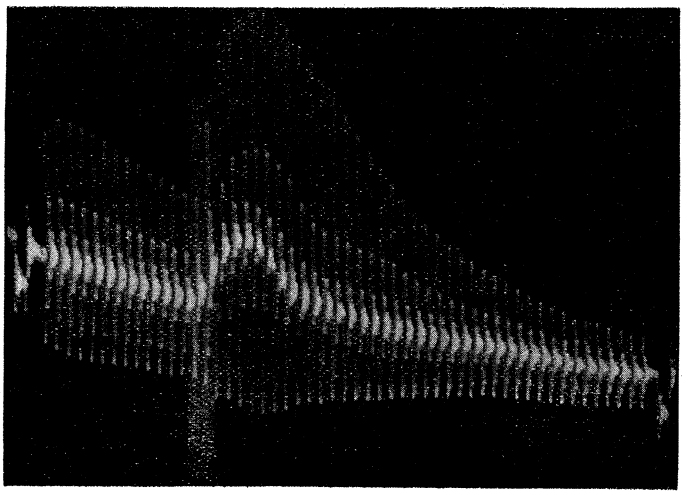
Figs. 17, 18, and 19. "Full," "medium," and "slight" effects of CO_2.

With stale nerve, kept in decinormal saline solution (0·58 per cent.) for 2 or 3 or more hours, the primary effect is less marked, and may only amount to a diminution short of complete abolition, the secondary effect is well marked.

(2). *The effect of a small amount of carbon dioxide upon normal nerve is a primary augmentation of excitability* (figs. 16, 19, 20).

Expired air, *e.g.*, containing 3 per cent. CO_2, produces primary augmentation (fig. 20).

Fig. 20. (Obs. 589.)



Effect of expired air.

(3). *The effect of a moderate amount of carbon dioxide is primary augmentation giving way to diminution and followed by secondary augmentation of excitability* (figs. 15 and 18).

The evident conflict taking place between movements in two opposite directions is in part due to an unequal action of CO_2 upon opposite processes in excited nerve. As will be shown later, CO_2 may, *at the same time*, give rise to augmentation of the negative deflection with diminution of the positive deflection.

The use of the indefinite terms "large," "moderate," and "small," is consequent upon the unavoidable variability in the physiological quality of excited nerve. A quantity of CO_2 that is large in relation to one nerve, giving the typical effect, may be moderate in relation to another nerve, or even small in relation to a third, giving primary augmentation. Such differences are partly connected with state of health, partly with the previous influences to which the nerves have been submitted, and partly with the mere bulk of nerve, in this last case by virtue of more gradual diffusion of the gas into thick than into thin nerves; the large nerves of German frogs, *e.g.*, required larger doses than the small nerves of English frogs to produce similar effects; on the other hand, the former gave remarkably distinct CO_2 effects in consequence of tetanisation. Strength of stimulation also influences the result; this

strength, although as previously stated, taken with uniform coil distance, must vary with thickness of nerve and distance between exciting poles, and if the nerve is very thick or the distance between the poles less than usual, 10 units may possibly prove too high a stimulation strength to allow of the demonstration of complete abolition of excitability.

Fig. 21. (Obs. 671.)

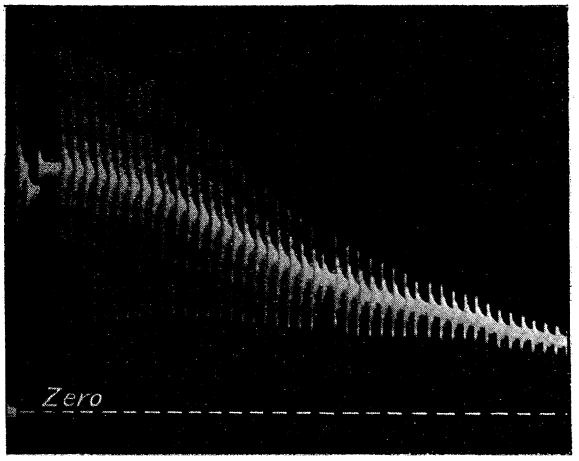
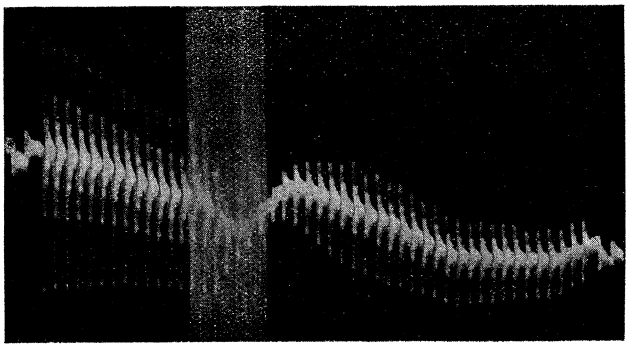


Fig. 22. (Obs 672.)

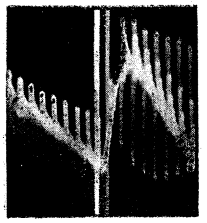


Records characteristic of nerve "carbonised" by prolonged sojourn in the living tissues of a pithed animal. The deflection is at first much larger than usual and declines more rapidly (fig. 21), the abolition caused by CO_2 is more prolonged than usual, and the after-augmentation is comparatively insignificant (fig. 22).

It is the excitability of the whole strand of protoplasm that is tested under the conditions of observation, the direct and the indirect or propagated molecular disturbance caused by stimulation, and modified by CO_2 or other gas. To determine whether the modification was more pronounced at the origin of the disturbance or at the part connected with the galvanometer, observations were taken with a gas-chamber carefully divided into two compartments, the nerve being passed through a

small hole in the dividing wall, and the division made good by moist saline clay round the nerve and blocking the hole. CO_2 was then passed through one or other compartment; in both cases the characteristic modifications of excitability were produced, more pronounced however when the gas was passed through the compartment containing the leading-off electrodes than when it was passed through that containing the exciting electrodes. Similar results occurred with ether vapour.

Fig. 23. (Obs. 788.)



Currents of action, and effect upon them of CO_2, with the exciting and galvanometer circuit conjoined (as described on pp. 12, 13).

§ 2. *Action of Carbon Dioxide on Modified Nerve.*

The description hitherto given is that of the action of CO_2 upon normal nerve, giving the usual current of injury and excitatory negative deflection.

We have now to describe the action of carbon dioxide upon abnormal nerve, giving little or no current of injury, and an excitatory deflection in a positive direction.

This description is a necessary preface to any consideration of the evidence to be offered of a production of CO_2 within the nerve itself, but must itself be prepared by a summary description of the various kinds of excitatory deflections manifested by more or less profoundly modified nerve under regular excitations of regular strength (10 units) and duration ($7\frac{1}{2}$ secs.).

Deferring to a later stage (p. 45) any theoretical consideration as to the possible nature of a positive deflection, and simply to render possible an orderly description of the action of carbon dioxide, the following classification of nerve effects is adopted.

(1). During a first stage (fresh nerve) the galvanometric response to excitation consists in a large negative deflection succeeded by little or no positive after deflection.

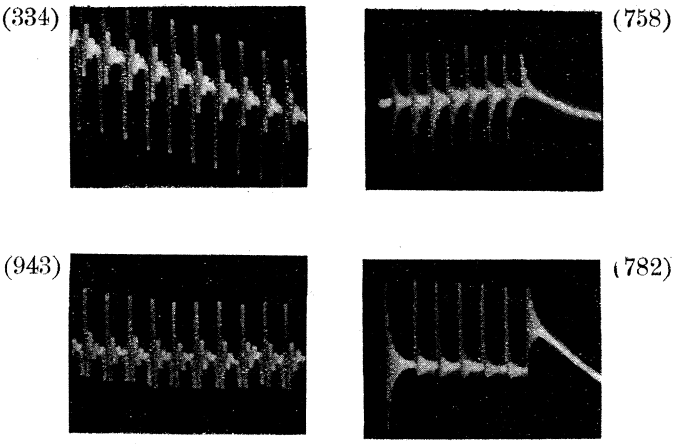
(2). During a second stage (transitional nerve) the galvanometric response consists in a small negative deflection succeeded by a large positive after-deflection.

(3). During a third stage (stale nerve) the response consists in a positive deflection followed by a positive (rarely by a negative) after-deflection.

This division is, of course, more or less arbitrary, the transition from stage to stage

is not sudden but gradual, as will be indicated more fully in Chapter IV., when we shall also inquire into the possible nature of positive deflections. For our present purpose the classification is indispensable.

Fig. 24.



Observations with the partially damped galvanometer, in 334 and 943, with an oscillation period of $12\frac{1}{2}$ secs., and a swing-back $= \frac{2}{3}$ the original swing; in 758 and 782, with an oscillation period of $8\frac{1}{2}$ secs., and a swing-back $= \frac{1}{3}$ the original swing.

No.	Effect.	Uncorrected after-effect.	Correction.	Corrected after-effect.
334	−10	+6·6	+6·6	0
943	− 4·5	+7	+3	+4
758	+ 6	−6	−2	−4
782	+12	−1	−4	+3

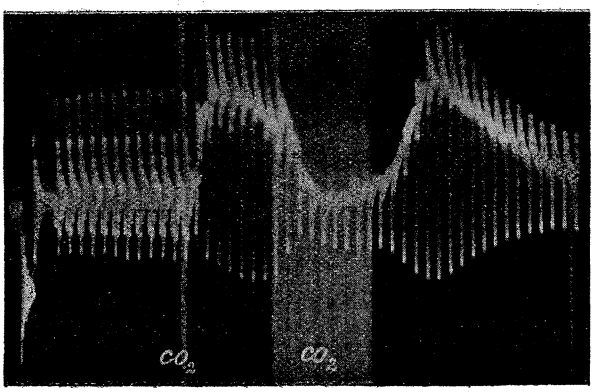
The action of CO_2 upon nerve in the first stage has been fully described above (p. 19).

The action of CO_2 upon nerve in the second stage is to augment the negative deflection and to diminish the positive after-deflection.

The action of CO_2 upon nerve in the third stage is to diminish the positive deflection or to substitute for it a negative deflection.

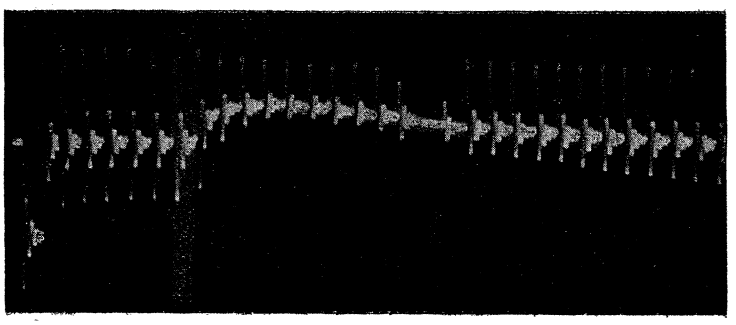
The data upon which these last two conclusions are based are contained in the following summary of observations.

Fig. 25. (Obs. 734.)



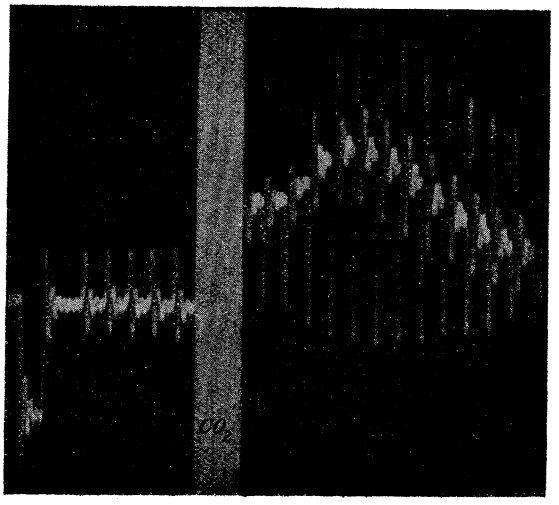
Influence of "little" and "much" CO_2 upon nerve in the second stage. (See Table B.)

Fig. 26. (Obs. 982.)



Influence of CO_2 upon nerve in the third stage; diminution of the positive effect.

Fig. 27. (Obs. 881.)



Influence of CO_2 upon nerve in the third stage; reversal of effect from positive to negative.

TABLE B.—Summary of Observations on the Action of Carbon Dioxide upon Modified Nerve.

Number.	Plate number.	CO_2.	
1	710 (fig. 35)	1 min. (150 cub. centims.)	The original deflection is a small S. effect, followed by a large N. after-effect. The first effect of CO_2 was to augment the S. and to diminish the N. Subsequently the S. and the N. were both increased.
2	711	10 mins. (1600 cub. centims.)	Same nerve. Similar results. The nerve had been kept for 18 hours in normal saline, and previously submitted to the action of ether vapour with a similar result, viz., simultaneous augmentation of S. and diminution of N. as the secondary result of etherisation.
3	727	2 mins. (600 cub. centims.)	Primary abolition followed by secondary augmentation, the augmentation of S. being greater than that of N. The nerve had been previously treated with HCl $n/40$ for 1 min.
4	734 (fig. 25)	100 cub. centims.	German frog. Nerve in normal saline for 3 hours. The original deflection is a small S. effect, followed by a large N. after-effect, giving place to a large S. followed by a small N., in consequence of the first dose of CO_2.
		1500 cub. centims.	The second and larger dose gives diminution of S. and abolition of N.
5	738	7 mins.	German frog. Nerve in normal saline for 15 minutes. Augmentation of S. with diminution of N.
6	742	7 mins. (twice)	Frog killed 3 hours; nerve left *in situ*. Primary effect { Slight diminution of S. effect. / Greater diminution of N. after-effect. Secondary effect { Rapid, but slight augmentation of S. / More gradual recovery of N.
7	743	4 mins.	German frog. Nerve for 4 hours in saline. Augmentation of S. with diminution of N., followed by augmentation of S. and slower augmentation of N.
8	744	4 mins.	English frog. Nerve 15 minutes in saline. Similar result to that of 743.
9	745	..	Second nerve of same frog. 1½ hours in saline. Similar result. N.B.—Ether subsequently gave a similar result.
10	747	.. 1 min. 3 mins.	English frog. Nerve 10 minutes in saline. Augmentation of N. and of S. 7 minutes later, diminution of S. and abolition of N.
11	752		Nerve in saline for 6 hours, and previously used for tetanus effect (751). The original deflection was s.N. The deflection after tetanus was S.N. The effect of CO_2 was primary abolition, followed by augmented S. before the reappearance of N.
12	786	1 min.	Primary augmentation of S. and N. S. is augmenting before N. N. is still augmenting when the augmentation of S. has begun to decline.
13	788 (fig. 23)	1 min.	Secondary coil and galvanometer in the same circuit. North deflection altered by CO_2 to a south deflection. Subsequently S. is diminishing while N. is increasing.
14	{ 837 / 838 }	1 min.	North deflection abolished, gradually returning. Nerve 4 hours after excision, previously used for urea.

TABLE B.—Summary of Observations on the Action of Carbon Dioxide upon Modified Nerve—(continued).

Number.	Plate number.	CO_2.	
15	839	2 mins.	North deflection gives way to small north followed by south after-deflection.
16	841 842	2 mins.	Nerve $8\frac{1}{2}$ hours in saline. North replaced by south deflection, and subsequently returning.
17	843 (fig. 41)	4 mins.	Same nerve. Similar result.
18	844	4 mins.	Same nerve. Similar result.
19	851	1 min.	Nerve $1\frac{1}{2}$ hours after excision, previously used for urea. Augmentation of the quotient S/N.
20	859 (fig. 31)	3 mins.	Nerve left for 6 hours in blood-serum, then used for another 6 hours for various experiments (CO_2, acetone vapour, tetanus). N.n. temporarily replaced by S.n. (The same nerve previously tested by 5 minutes' tetanisation gave a similar result; Obs. 858, fig. 30).
22	871	1 min.	1 hour after excision. Typical increase by "little" CO_2 in expired air. The same nerve 8 hours later gave a north deflection that was diminished by 5 minutes' tetanus (see Obs. 874, fig. 45).
23	877	1 min.	Conversion of s/N into S/n.
24	881 (fig. 27)	2 mins.	Nerve left for 15 hours in saline. Replacement of N. by S.
25	882	2 mins.	Same nerve. Diminution of N.
26	886	2 mins.	Nerve left for several hours in saline. Conversion of s/N into S/n.
27	935	4 mins.	Nerve left for 10 hours in saline. Variation s/N. Augmentation of s. with diminution of N., followed by diminution of s., and subsequently by augmentation, *i.e.*, typical effect of CO_2 in "moderate" amount.
28	982 (fig. 26)	1 min.	Nerve 2 hours after excision, previously used for creatine; diminution of N. deflection.
29	983	4 mins.	Same nerve. Abolition of N. deflection. Reappearance of S., subsequently S.N., s.N., n.N. (The same nerve shortly afterwards gave the original deflection N., which was diminished by 5 minutes' tetanisation (Obs. 984, fig. 32). An hour later the north variation was reversed by tetanisation for 13 minutes (Obs. 986).
30	990	1 min.	Nerve 2 hours after excision; previously tested by CO_2, and by sarcosin. Diminution of N. deflection. (The same nerve previously tested by 5 minutes' tetanisation, exhibited diminution of the N. deflection (Obs. 989).
31	992	1 min.	Nerve kept for $3\frac{1}{2}$ hours in saline, and previously used for tetanus (991) giving a *s.* deflection, followed by a N. after-deflection. Subsequently to CO_2 the s. deflection is greatly augmented, the N. after-deflection is relatively diminished.
32	993	1 min.	Same nerve about half hour later gives o.N. deflection and after-deflection, converted to S.N. by CO_2. (See Obs. 991 and 994 in Table C.)
33	2006	1 min.	Nerve 6 hours in saline. Variation s.N. before CO_2, which produced diminution of s., followed by augmentation, and still greater diminution of N. (The same nerve, previously tested by 5 minutes' tetanisation, exhibited a similar temporary diminution of s.; Obs. 2005).

TABLE B.—Summary of Observations on the Action of Carbon Dioxide upon Modified Nerve—(continued).

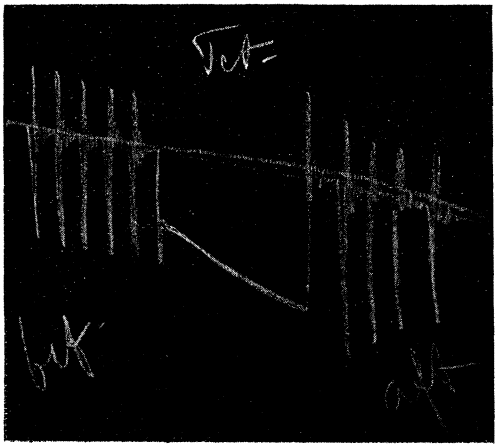
Number.	Plate number.	CO_2.	
34	2067	1 min.	Nerve 30 hours in saline (Jan. 11, 1896). N. temporarily replaced by S. deflection. (The same nerve, previously tested by 5 minutes' tetanisation, gave only a diminution of N.; Obs. 2066; in other respects the two records are precisely similar.)
35	2073	2 mins.	Nerve kept for 2 hours in 5 per cent. lactose solution, and giving a N. deflection, which is reversed to S. by CO_2. (The same nerve gave a similar reversal in consequence of 5 minutes' tetanisation; Obs. 2072.)
36	2075	2 mins.	Same nerve; similar result, viz., reversal (Obs. 2074 gave similar result by 5 minutes' tetanisation of the same nerve).
37	2080	2 mins.	Nerve kept for 4 hours in lactose solution, 5 per cent., and giving a deflection s.N. Augmentation of *s.* in consequence of CO_2. (The same nerve gave a similar but less marked effect, viz., augmented s., in consequence of 5 minutes' tetanisation.)
38	2082	2 mins.	Nerve kept for 8 hours in saline, giving a N. deflection, which is temporarily reversed to S. after CO_2. (The same nerve gave an augmentation of the N. deflection after 5 minutes' tetanisation; Obs. 2081.)
39	2241	..	Dead-beat galvanometer. Nerve kept for $\frac{3}{4}$ hour in saline. Great augmentation of S. deflection; subsequent appearance of N. after-deflection.
40	2242	..	Same nerve continued. The response is now s.N., and is temporarily changed to S.o. by CO_2.
41	2248	..	Dead-beat galvanometer. Nerve in saline for $\frac{1}{4}$ hour. Great augmentation of S. deflection by CO_2.
42	2249 (fig. 67)	..	Dead-beat galvanometer. Nerve in saline for $1\frac{3}{4}$ hours. Great augmentation of S. deflection, subsequent appearance of N. after-deflection.
43	2251	1 min.	Dead-beat galvanometer. Fresh nerve. Original effect S.n.; augmentation of S. and abolition of n. by CO_2, subsequently augmentation of n.
44	2253	..	March 12, 1896 (Lecture experiment). Original variation s., augmented by little CO_2; augmentation subsequently abolished by more CO_2 and followed a few minutes later by secondary augmentation.
45	2256	3 mins.	Dead-beat galvanometer. Experiment made twice at interval of 10 minutes. In each case primary diminution of S. deflection followed by secondary augmentation.
46	2257	..	Dead-beat galvanometer. Nerve left *in situ* for 12 hours before excision. Primary diminution of S., not followed by secondary augmentation.
47	2268	2 mins.	Dead-beat galvanometer. Nerve in saline for 1 hour. Original effect s.n.; primary abolition, secondary augmentation.
48	2274	1 min.	Dead-beat galvanometer. In saline 4 hours; fresh transverse section. Primary augmentation of S deflection only, soon followed by augmentation of n. after-deflection.
49	2276	1 min.	Same nerve; 2 hours later the deflection is n., temporarily abolished and reversed to s. by CO_2.
50	2277	1 min.	The same nerve, 15 hours later, gives a deflection s. (without new transverse section, diminished by CO_2).

Chapter III.—Production of CO_2 in Tetanised Nerve.*

§ 1. *Evidence in Normal Nerve.*

Evidence of chemical and thermic change in active nerve, analogous with that obtained in the case of active muscle, does not as yet exist. That now offered of a production of CO_2 within isolated nerve rests upon our knowledge of the effects produced upon nerve by CO_2 reaching it from without. It will be shown in the present communication that tetanised nerve manifests alterations identical in kind with the alterations characteristic of the action of CO_2. It is, perhaps, permissible to state that the alterations were forecast by the following argument and diagram (fig. 28) before they were verified by experiment (fig. 29).

Fig. 28.



Hypothesis.

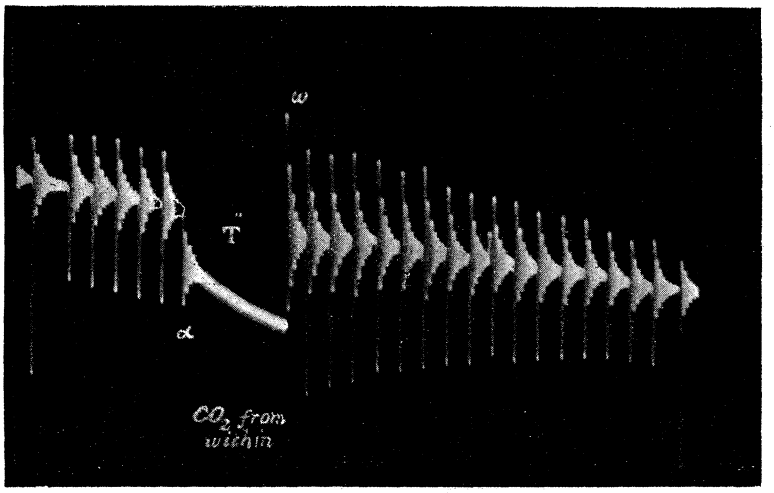
CO_2 in very small quantity produces an augmentation of the negative, or south deflection, and the delicacy of the reaction is extreme. If, then, tetanisation of nerve is attended with any CO_2 production, we may expect that such CO_2, arising within the nerve itself, will effect the characteristic augmentation. The negative deflection should be greater after a prolonged tetanus, the augmentation should gradually diminish with the presumable dissipation of CO_2; moreover, the negative

* In view of the criticism to which the above title has given rise, to the effect that no carbon dioxide has been separated and identified, it appears desirable, if somewhat superfluous, to premise that no CO_2 gas has been collected and identified by chemical tests, and that the evidence offered rests entirely upon the physiological test afforded by the nerve itself in various states; the nerve serves as the reagent, reacting in all particulars to tetanisation as it does to CO_2, from which, as in the case of any other test (with more or less certainty, according to the circumstances under which the test is made), the inference is drawn that CO_2 is produced during tetanisation, on the principle that, under similar circumstances, similar effects are due to a similar cause.

deflection of the tetanus itself should increase with the presumable evolution of CO_2 during its progress—as in the diagram (fig. 28).

The forecast was borne out in all its features, as may be seen in fig. 29.

Fig. 29. (Obs. 675.)



Verification.

§ 2. *Evidence in Modified Nerve.*

Confirmatory evidence towards the same conclusion is afforded by the following complementary experiments upon nerve in the second and third stages.

As has been shown above (p. 25), the typical effect of CO_2 upon a reversed excitatory effect (north or positive) of the third stage is to restore that reversed effect to its original character as a (south) negative deflection.

Tetanisation effects a similar reversal of the positive effect.

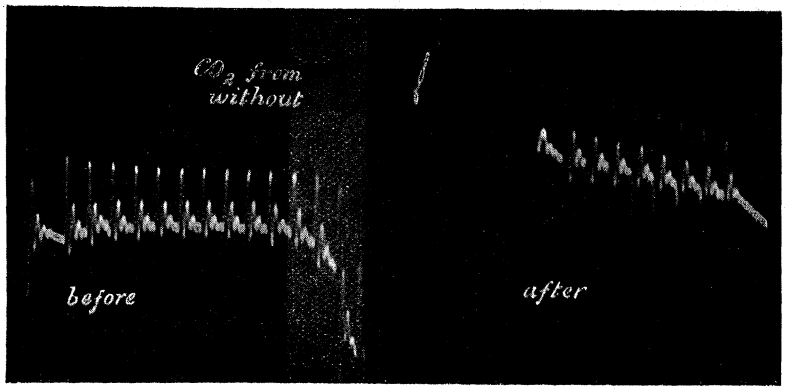
Fig. 30 exhibits this reversal of a positive (north) deflection, and its replacement by a negative (south) deflection, in consequence of CO_2, passed through the gas-chamber.

Fig. 31 exhibits upon the same nerve, under similar conditions, the similar effect of tetanisation, presumably by reason of CO_2 evolved within the nerve itself.

In addition to these two representative experiments, two further experiments, which, however, are no more than varieties of the second, afford corroborative evidence.

As has been stated above (p. 25), the effect of carbon dioxide upon the north or positive deflection of the third stage does not always amount to the complete reversal of deflection, but only to its diminution; a similar effect may then be produced by tetanisation. The apparently contradictory nature of this result as compared with that obtained on nerve of the first stage, viz., diminution of an effect of excitation, is in reality confirmatory evidence; in this, as in the other cases, a negative sum or process has been augmented (or a positive sum or process has been diminished) as an effect of CO_2 applied from without or arising from within.

Fig. 30. (Obs. 859.)



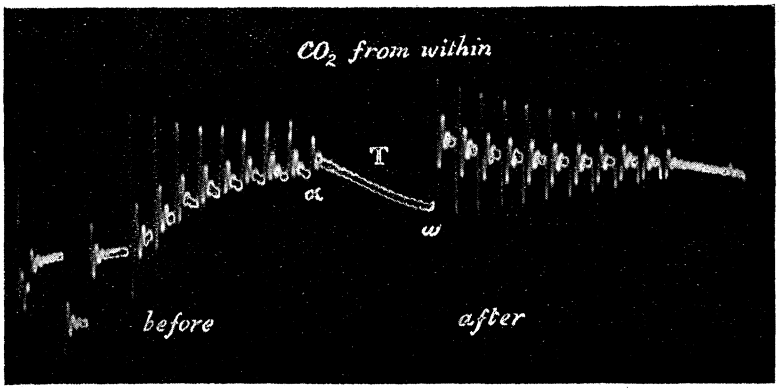
Nerve in third stage after half an hour's rest. Influence upon the deflection of a slow stream of CO_2.

Before CO_2.—The deflections are north north, consisting of a positive effect followed by a positive after-effect.

During the passage of CO_2 the spot fell off plate south. Subsequently, having been readjusted north, it rose off plate north.

After CO_2.—Large negative effect diminishing, smaller positive after-effect increasing. Half an hour later the deflections were again north north.

Fig. 31. (Obs. 858.)



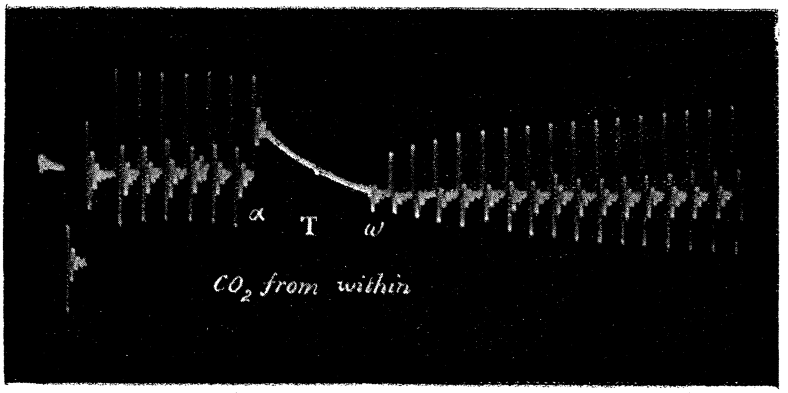
Same nerve giving a positive effect, followed by a positive after-effect. North north.

Before Tetanus.—Positive effects rapidly diminishing. Positive after-effects summating and causing the base line to rise. The last three effects exhibit a slight negative effect at the end of each excitation, interposed between the positive effect and the positive after-effect.

During Tetanus.—Positive deflection becoming increasingly negative. The first deflection at the commencement of the tetanus is north, the first deflection at the end of the tetanus is north.

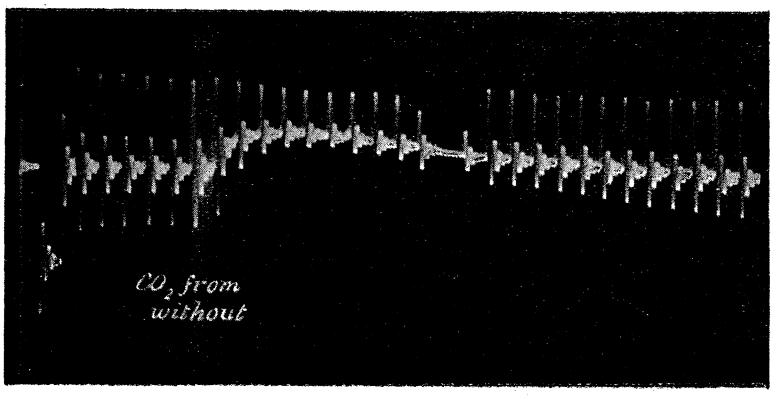
After Tetanus.—Large negative effects diminishing, smaller positive after-effects increasing. After a subsequent half hour of repose, the negative effects had vanished, the deflection had again become north north.

Fig. 32. (Obs. 984.)



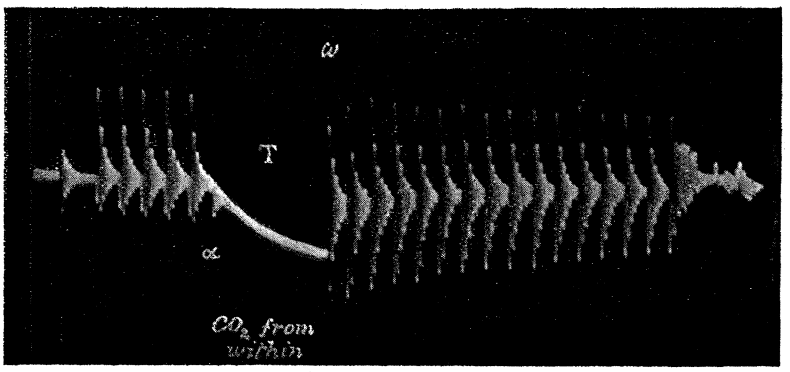
Influence of tetanisation upon nerve in the third stage; diminution of the positive effect.

Fig. 33. (Obs. 982.)



Influence of carbon dioxide upon nerve in the third stage; diminution of the positive effect. (The irregularity of response from the 16th to 18th minute is due to irregularity of the stimulating apparatus.)

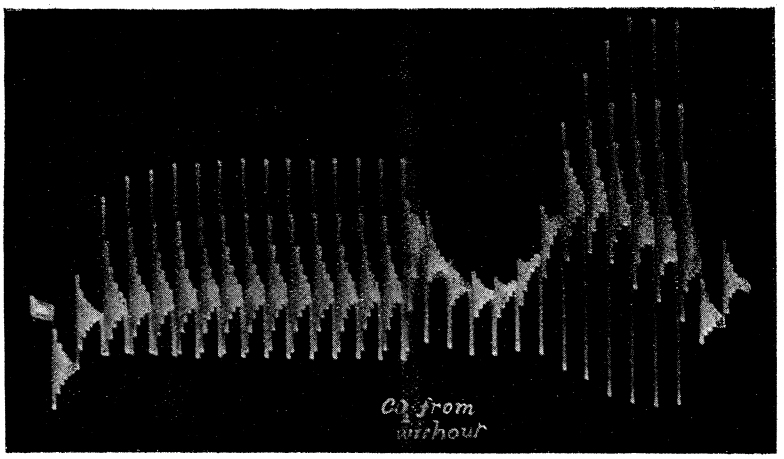
Fig. 34. (Obs. 762.)



Influence of tetanisation upon nerve in the second stage, giving the response s.N., viz., small negative effect followed by large positive effect; augmentation of the negative effect.

When further, as in the case in the second stage, the effect of CO_2 upon a small negative or south deflection followed by a large positive or north after-deflection, is to reverse the relative magnitudes of the two deflections, a similar effect is produced by tetanisation.

Fig. 35. (Obs. 710.)



Influence of carbon dioxide upon nerve in the second stage, giving the response *s*.N.; augmentation of the negative effect.

There is, in short, complete similarity between the effects of external CO_2 in various states of nerve and those of the presumably internal CO_2 of tetanisation during these same various states; a correspondence so close, that the evidence may be held as conclusive. Whether disintegration products other than CO_2 are produced by nerve-tetanisation is of course neither affirmed nor denied.*

The accompanying summary (Table C) contains the experimental data upon which the general conclusion is based that CO_2 is produced in tetanised nerve.

* As will be shown in Chapter IV., the principal effect of CO_2 upon anelectrotonic and katelectrotonic currents is augmentation, a similar effect is produced by prolonged tetanisation. But here, as in the case of the negative variation, quantity of CO_2 in relation to state of nerve has to be taken into account, and my experiments in this direction are not sufficient to justify categorical statements.

TABLE C.—Summary of Data in Evidence of the Production of Carbon Dioxide in Normal and in Modified Nerve.

Under the heading "Character of the Deflection" is also given, when possible, that of the after-deflection.
Under the heading α is given the deflection at the beginning of the prolonged tetanus, under the heading ω that at its end.
Numerical measurements of several of the observations in this table, as well as in Table B, are given in Table D.
The time of year at which observations were taken may be ascertained by reference to the "time-table" at the end of the paper.

No.	Plate No.	Conditions of experiment.	Duration of tetanisation.	Character of deflection before tetanisation.	α.	During tetanisation.	ω.	Character of deflection after tetanisation.	Remarks.
			minutes.						
1	675 (fig. 29)	Nerve in saline for 1 hour after excision	5	S.N.	S.	Increasing	N.	S.N.	Table D.
2	676	A few minutes after excision . .	10	S.N.	S.	,,	N.	S.N.	
3	677	Nerve left in pithed frog for 1 hour before experiment	10	S.N.	S.	,,	N.	S.N.	Declining normal.
4	678	Same nerve as in 676; nerve put aside for 1 hour in saline	10	S.N.	S.	,,	? S.	S.N.	
5	679	A few minutes after excision . .	10	S.N.	S.	,,	N.	S.N.	Declining normal, more especially as regards the N. after deflection.
6	680	Nerve in saline for 2 hours after excision	10	S.N.	S.	,,	? S.	S.N.	
7	689	Nerve in saline for $\frac{3}{4}$ hour after excision	20	S.N.	S.	Increasing S.	? S.	S.N.	Augmented deflection S., diminished after deflection N., subsequent to tetanisation.
8	737	2 hours in saline; previously used for ether experiment (735)	5	S.N.	..	,,	...	S.N.	
9	741	1 hour in saline; previously tested by CO_2 (740)	5	S.N.	S.	,,	N.	S.N.	Typical effect of CO_2 in Obs. 740 on same nerve.
10	751	6 hours in saline	5	n.N.	..	Decreasing N.	..	S.N.	Reversal. Typical effect of CO_2 in Obs. 752 on same nerve.
11	758	$5\frac{1}{2}$ hours in saline	5	N.S.	N.	,,	s.N.	s N.	Reversal from N. to s.
12	761	4 hours in saline; previously tested by oxalic acid n/20	5	S.	S.	Increasing S.	N.	S.	Augmentation of S.
13	762 (fig. 34)	6 hours in saline	5	s.N.	s.	,,	N.	S.N.	Augmented S.

TABLE C.—Summary of Data in Evidence of the Production of Carbon Dioxide in Normal and in Modified Nerve—(continued).

No.	Plate No.	Conditions of experiment.	Duration of tetanisation.	Character of deflection before tetanisation.	α.	During tetanisation.	ω.	Character of deflection after tetanisation.	Remarks.
			minutes.						
14	782	10 minutes in saline, then heated to 41°, then record taken	5	N.N.	N.	Decreasing N.	S.	N.	The original deflection before heating was S.n. (Obs. 781).
15	857	$6\frac{1}{2}$ hours in serum; previously tested by acetone vapour	5	N.N.	s.	„	N.	s.N.	Acetone vapour had had no apparent effect (Obs. 855).
16	858 (fig. 30)	Same nerve after $\frac{1}{2}$ hour rest . .	5	n.N.	n.	„	N.	s.N.	
17	862	3 hours in saline; previously tested by urea 2 per cent. solution	5	s.N.	s.	Increasing S.	N.	S.N.	
18	863	Same nerve	5	s.N.	s.	„	N.	S.N.	
19	865	„ „	5	s.N.	s.	„	N.	S.N.	
20	866	„ „	5	s.N.	s.	„	N.	S.N.	
21*	873	3 or 4 hours in saline; previously used for ether experiment (870)	5	N.	N.	Decreasing N.	S.	N.	
22	874 (fig. 45)	Same nerve	5	N.	N.	„	S.	N.	
23	875	In saline a few minutes	5	S.	S.	Increasing S.	N.	S.	
24*	876	Same nerve	5	S.	S.	„	N.	S.	Repeated twice; increased S. after the first tetanus, decreased S. after the second. CO_2 subsequently gave marked increase.
25*	878	In saline 13 hours	7	N.	N.	Decreasing N.	S.	N.	
26	880	In saline 1 hour	5	o.N.	o.	Increasing S.	n.	S.N.	Reversal.
27	882	Same nerve 1 hour later. . . .	5	N.	N.	Decreasing N.	S.	N.	Very little alteration by tetanus, and only diminution of N. by CO_2.
28*	883	Same nerve	5	S.N.	S.	Increasing S.	n.	s.	
29*	885	„ „	5	N.	N.	Decreasing N.	S.	N.	Repeated twice; with augmentation of N. in each case.
30	898 (fig. 46)	Nerve previously tested by sarcosin solution 1 per cent. (897)	5	N.	N.	„	N.	S.N.	Reversal.

TABLE C.—Summary of Data in Evidence of the Production of Carbon Dioxide in Normal and in Modified Nerve—(continued).

No.	Plate No.	Conditions of experiment.	Duration of tetanisation.	Character of deflection before tetanisation.	α.	During tetanisation.	ω.	Character of deflection after tetanisation.	Remarks.
			minutes.						
31*	905 (fig. 36)	Nerve previously tested by creatin solution 2 per cent. (902–3)	10	N.	N.	Decreasing N.	S.	N.	Augmented N. Obs. 906 with CO_2 on the same nerve is also exceptional.
32	912 (fig. 37)	In saline 15 hours.	10	s.N.	s.	Increasing S.	N.	s.n.	Diminished s., also diminished after-effect N.
33	928	Nerve previously tested by muscle extract (926, 927)	5	S.N.	S.	,,	N.	S.N.	Great augmentation of S. See Table D.
34	929	Same nerve	5	s.n.	s.	,,	N.	S.N.	Great augmentation of S. See Table D.
35	934	In saline 10 hours; previously tetanised for $1\frac{1}{2}$ hour	5	S.N.	S.	,,	N.	S.N.	No appreciable effect; on the same nerve CO_2 for several minutes gave only augmented S.
36	937	In saline 24 hours	5	S.N.	S.	,,	N.	S.N.	Augmented S.
37	940	,, 14 ,, ...	5	S.N.	S.	,,	N.	S.N.	Slight augmentation of S.
38	941	,, $1\frac{1}{2}$,, ...	30	S.N.	S.	,,	N.	S.N.	Augmented S.
39*	942	Same nerve	5	N.	N.	Decreasing N.	S.	N.	Augmented N.
40	946	In saline 3 hours; previously used for serum-albumin (945)	5	s.N.	s.	Increasing S.	N.	S.N.	Augmented S.
41	950	Previously used for diabetic sugar (949)	5	n.s.	n.	Decreasing N.	S.	n.s.	Diminished N.
42	954	2 hours after excision; previously used to test diabetic sugar (951–2–3)	5	n.s.	n.	,,	S.	n.s.	Diminished N.
43	955	Same nerve	5	N.S.	N.	?	?	n.s.	Diminished n. deflection; less, if at all, diminished S. after-deflection.
44	958 (fig. 39)	In saline 5 hours; previously used to test diabetic sugar (956–7)	5	s.N.	S.	Increasing S.	N.	S.n.	
45	960 (fig. 42)	In saline 10 hours; previously used for diabetic sugar	5	o.N.	O.	,,	N.	S.n.	
46	984 (fig. 32)	Previously tested by creatin 2 per cent. and by CO_2	5	N.	N.	Decreasing N.	s.	n.	Compare with Obs. 982, fig. 33.

TABLE C.—Summary of Data in Evidence of the Production of Carbon Dioxide in Normal and in Modified Nerve—(continued).

No.	Plate No.	Condition of experiments.	Duration of tetanisation.	Character of deflection before tetanisation.	α.	During tetanisation.	ω.	Character of deflection after tetanisation.	Remarks.
			minutes.						
47	985	Same nerve	5	N.	N.	Decreasing N.	s.	n.	Similar effect, viz., diminution of N.
48	986	Nerve 14 hours in saline . . .	12	N.	N.	,,	N.	S.	Reversal.
49	989	Previously tested by sarcosin 5 per cent.	5	N.	N.	,,	?	n.	Diminution of N. The same nerve gave similar diminution by CO_2. Obs. 990.
50	991	$2\frac{1}{2}$ hours in saline	5	s.N.	s.	Increasing S.	N.	S.N.	Augmentation of S. Similar result on the same nerve by CO_2; Obs. 992; about 1 hour later o.N. deflection altered to S.n. by CO_2; Obs. 993. This series, 991–2–3–4, illustrates very plainly the identical results of tetanus and of CO_2.
51	994	Same nerve	5	o.N.	o.	,,	N.	s.N.	
52	2003	2 hours in saline; previously tested by propionic acid, n/20	5	S.	S.	,,	n.	S.	
53	2004	6 hours in saline	5	s.N.	s.	?	N.	S.N.	
54*	2005	Same nerve	5	S.N.	S.	Increasing S.	N.	s.n. S.N.	Diminution of S. followed by augmentation. Similar effect by CO_2 in next observation on same nerve: Obs. 2006.
55*	2030	In saline 18 hours; previously tested by propionic acid, n/50 and n/40 (2026–7)	5	S.S.	S.	,,	? N.	S.N.	CO_2 on the same nerve subsequently gave the usual effect; Obs. 2031.
56	2054	In saline 19 hours (Jan. 12, 1896)	5	S.	S.	,,	N.	S.	Augmentation of S.
57	2055	Same nerve	5	S.	S.	,,	N.	S.	Augmentation of S.
58	2057	Same nerve	5	S.	S.	,,	N.	S.	Augmentation of S.
59	2060	Same nerve left in saline 12 hours longer	5	S.	S.	,,	N.	S.	Augmentation of S.
60	2065	In saline 26 hours (Jan. 13, 1896)	5	N.	N.	Decreasing N.	N.	n.	Diminution of N.

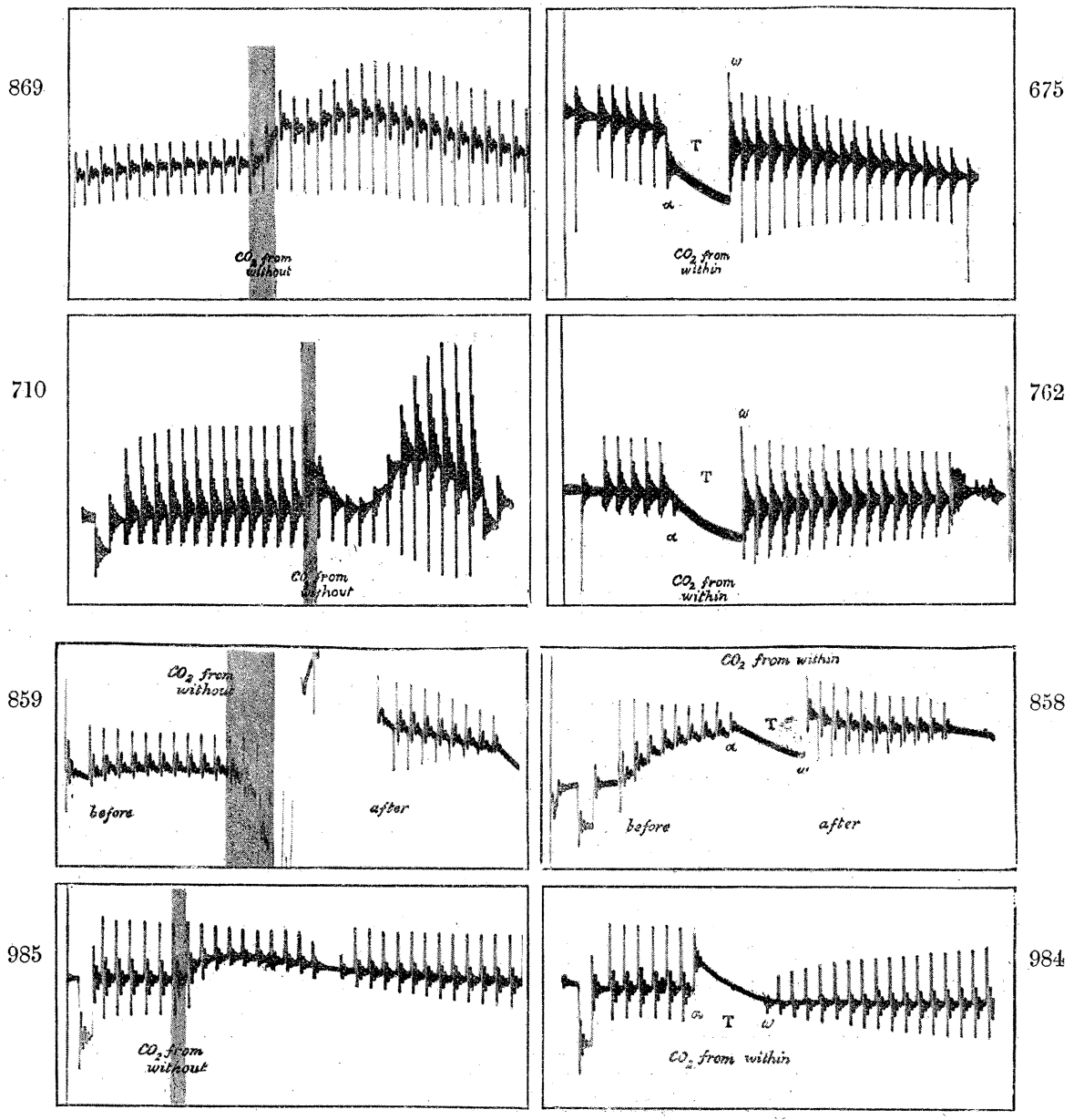
TABLE C.—Summary of Data in Evidence of the Production of Carbon Dioxide in Normal and in Modified Nerve—(continued).

No.	Plate No.	Conditions of experiment.	Duration of tetanisation.	Character of deflection before tetanisation.	α.	During tetanus.	ω.	Character of deflection after tetanisation.	Remarks.
			minutes.						
61	2066	Same nerve	5	N.	N.	Decreasing N.	N.	n.	The same nerve, subsequently tested by CO_2, gave reversal to s. soon followed by return to n. and N.; Obs. 2067.
62	2072	After lactose 5 per cent. for $1\frac{3}{4}$ hour	5	n.N.	n.	,,	N.	s.N.	Reversal. Similar effect by CO_2 in Obs. 2073.
63	2074	9 hours in saline	5	n.N.	n.	,,	N.	S.N.	Reversal. Similar effect by CO_2 in Obs. 2075.
64	2079	After 4 hours in lactose solution 5 per cent.	5	S.N.	S.	Increasing S.	N.	S.N.	Augmentation of S. Similar effect by CO_2 in Obs. 2080.
65*	2081	8 hours in saline	5	N.	N.	?	?	N.	Augmentation of N. CO_2 gave reversal from N. to S.; Obs. 2082.
66*	2084	Same nerve as in 2079, left in saline 18 hours longer; new transverse section	5	S.N.	S.	Decreasing S.	N.	s. Increasing.	Diminution of S. (The strength of tetanisation was 100 units.)
67	2258	10 minutes in saline	5	S.n.	s.	Increasing S.	N.	S.n.	S. deflection increased; n. after-deflection diminished. Dead-beat galvanometer.
68	2262	24 hours in saline; new transverse section	5	S.n.	S.	Steady deflection	N.	S.n.	S. deflection increased; n. after-deflection diminished. Dead-beat galvanometer.
69	2271	1 hour in saline	5	S.n.	S.	Increasing S.	N.	S.n.	Augmented S.; hardly any alteration of the after-deflection n. Dead-beat galvanometer.

NOTE.—In several of the above observations the same nerve has been tested by tetanisation and by CO_2 without removal from the electrodes or other disturbance; this has been the case, *e.g.*, in 740–741, 751–752, 858–859 (figs. 30–31), 982–984 (figs. 32–33), 989–990, 2005–2006, 2072–2073, 2074–2075, 2079–2080, 2081–2082.

On review of the foregoing data, it will be seen that the great majority of experiments gave clear and unambiguous results, but that in some (indicated by an asterisk in Table C) the results have been apparently ambiguous. These require commentary.

Fig. 35A.



Summary of observations presenting typical effects of CO_2 and of tetanisation upon nerve in the three states alluded to in text.

Action of CO_2 (869) and of tetanisation (675) upon nerve of the 1st stage.
" " (710) " " (762) " " 2nd stage.
" " (859) " " (858) " " 3rd stage, "clear effect."
" " (985) " " (984) " " " "disguised effect."

§ 3. *Ambiguous Phenomena in Modified Nerve.*

The typical and regular result with a fresh nerve (giving a large negative deflection during excitation) is as represented by Obs. 741 (fig. 29), an augmentation of the negative deflection. And as regards the tetanus itself, the deflection at its commencement is negative, at its end positive.

Similar results are obtained with a nerve in what has been termed a transitional state, neither "fresh" nor "stale" (giving little or no negative deflection followed by a large positive after-deflection); the negative deflection is increased and the positive after-deflection less increased, or diminished. With "stale" nerve (giving a positive deflection followed by a positive or a negative after-deflection) the results vary.

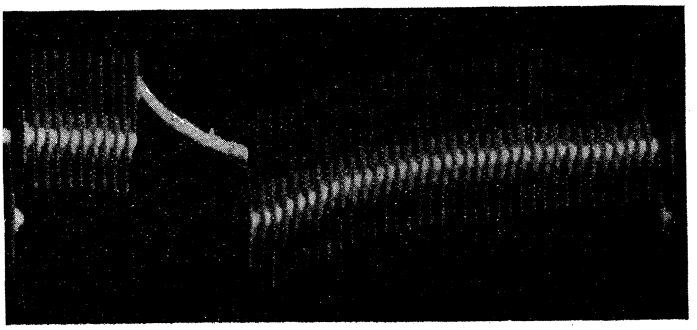
a. The deflection at the beginning of tetanus is positive and that at the end of the tetanus is positive, in which case a positive deflection before tetanus is replaced by a negative deflection after tetanus. This is the case that may be characterized as giving a "clear" effect, viz., reversal from N. to S., in consequence of CO_2 and of tetanisation.

b. The deflection at the beginning of tetanus is positive, at its end negative, in which case a positive deflection before tetanus remains as a diminished positive deflection after tetanus. This is the case that has been characterised as giving a "disguised" effect of CO_2 and of tetanisation, inasmuch as it presents an apparent exception to the rule that CO_2 augments the excitatory deflection. But even in this in some other respects anomalous case of "stale" nerves, the result may be recognized as offering no exception to the rule that CO_2 augments the negative deflection (or diminishes the positive deflection).

A noteworthy point in connection with the long tetanic deflection is that it always (at the stimulation strength of 10 units) progressively augments. It has only been with excessive strength of stimulation (100 units) that an augmentation has given way to diminution.

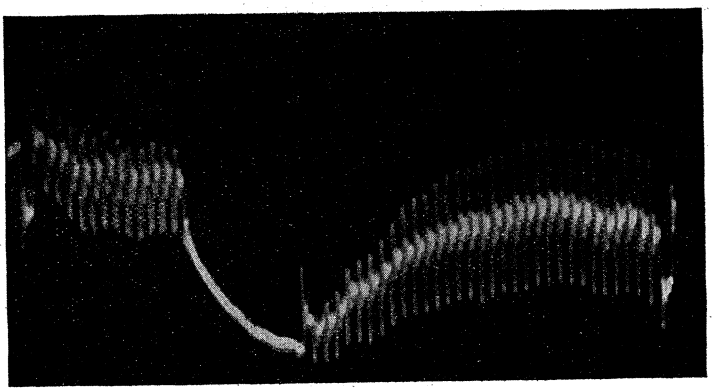
Occasional exceptions are met with to the rule that after the prolonged tetanus (at 10 units) a south variation is increased, or a north variation diminished; these have been distinguished in the table of data by an asterisk, and are illustrated in figs. 36 and 37. The possible significance of these exceptions where there is diminution of a south deflection, is indicated by the fact that a similar diminution can sometimes be demonstrated by using stimulation of excessive strength (fig. 38). Whether this diminution is to be regarded as a more profound effect of CO_2 in larger quantity, or as a fatigue effect, must be left an open question. The fact that the exceptional result may appear in the form of an augmented north deflection seems to be opposed to the latter alternative, and the key to these, in any case infrequent, peculiarities will probably be found in the peculiar differences of action of CO_2 in various quantities upon the deflection and after-deflection of nerve in various states; 905 exhibits this exceptional effect on a north variation, 912 on a south variation, and 2068 exhibits the dependence of the latter upon strength of tetanisation.

Fig. 36. (Obs. 905.)



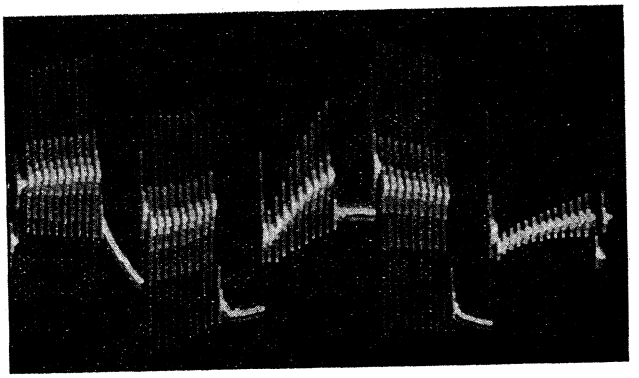
Nerve of the third stage. Exceptional effect of prolonged tetanisation. Augmentation of the positive deflection.

Fig. 37. (Obs. 912.)



Nerve of the second stage. Exceptional effect of prolonged tetanisation. Diminution of the negative deflection.

Fig. 38. (Obs. 2068.)



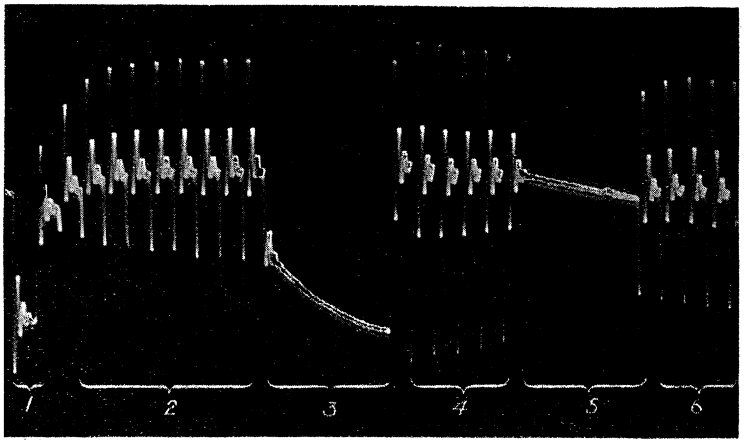
T 10↑ T 100↑ T 10↓ T 100↓

Nerve of the second stage. Augmented negative effect in consequence of weak tetanisation (10 units, both directions of coil). Diminished negative effect in consequence of strong tetanisation (100 units, both directions of coil).

§ 4. *The Staircase Effect.*

Under the conditions of these observations, isolated nerve nearly always exhibits the "staircase phenomenon" familar to physiologists in the contractile tissue of Medusa, in the Frog's heart, and in skeletal muscle. If the record of a normal series of deflections is commenced without preliminary testing, each deflection slightly (in

Fig. 39. (958.)



Nerve 5 hours after excision giving a deflection of the second stage. Excitation once a minute for $\frac{1}{8}$ minute with coil at 10 units. Tetanisation for 5 minutes from α to ω.

Before the tetanus the successive negative variations increase slightly forming a "staircase." After the tetanus the negative variations are increased, gradually diminishing. An interruption (*int.*) of all excitat ion shows how the diminution was delayed by the excitation itself.

		Presumable Change of CO_2.
1	Deflection by 0·001 volt	
2	Normal series at minute intervals exhibiting staircase increase	Evolution in small quantities at each tetanus.
3	Tetanus lasting 5 minutes . . .	Evolution in considerable quantity by long tetanus.
4	Normal series	Dissipation of CO_2 evolved during previous tetanus, in spite of short tetani.
5	Interruption of series by 5 minutes rest	More rapid dissipation of CO_2 effect while the latter are intermitted.
6	Normal series	

some cases markedly) exceeds its predecessor; the increment progressively diminishing so that a line passing through the culminating points is concave to the line of rest, and the S. : N. quotient gradually increases. In the case of nerve the phenomenon is in lesser degree a tetanus effect, similar to that described and considered above, viz., presumably a CO_2 effect; in place of the single large effect of a tetanus lasting several minutes, we are in presence of a gradually developing and summating series of smaller effects of tetani lasting only $\frac{1}{8}$ minute at intervals of a minute.

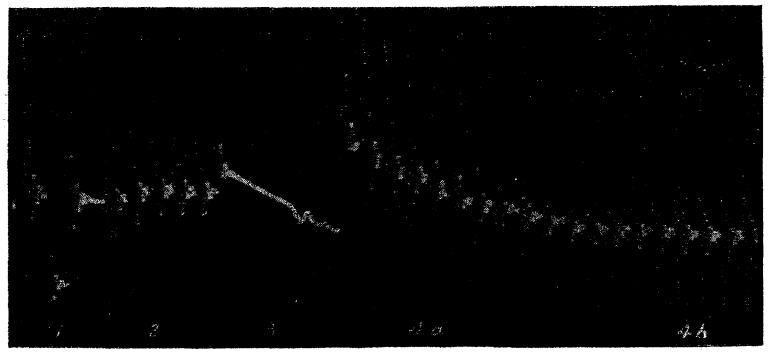
The regular effect of CO_2 is to increase the negative deflection of nerve in the first and second stages, and to diminish the positive variation of the third stage. The regular effect of tetani of $\frac{1}{8}$ minute at 1 minute intervals is to give series of slightly increasing negative deflections of nerve in the first stage, of markedly increasing negative deflections, and diminishing positive after-deflections of nerve in the second stage, and of diminishing positive deflections of nerve in the third stage (see fig. 31). In upwards of 500 observations available for study of that point no single exception is to be found to this rule, which is variously exemplified in all sorts and conditions of nerve. In the present communication it is best illustrated by the first portions of Obs. 958, fig. 39 (progressive increase of negative deflections of nerve in the second stage) of Obs. 858, fig. 31 (progressive diminution of positive deflections of nerve in the third stage), and of Obs. 2177, fig. 66.

Progressive Diminution of Positive After-Deflections of Nerve in the Second Stage.

Fig. 40 (Obs. 933) and fig. 41 (Obs. 843) are given in illustration of the temporary character of the reversal by tetanisation and by carbonic acid respectively. In the former case the reversal caused by 5 minutes tetanisation has disappeared in about 10 minutes; in the latter the reversal caused by a stream of CO_2 lasting for 5 minutes (*i.e.*, "much" CO_2) has endured for about 30 minutes.

Fig. 42 (Obs. 960) illustrates the great sensitiveness of nerve to tetanisation (and to CO_2) in the second stage; in this instance (which is one of the best marked of its

Fig. 40. (933.)



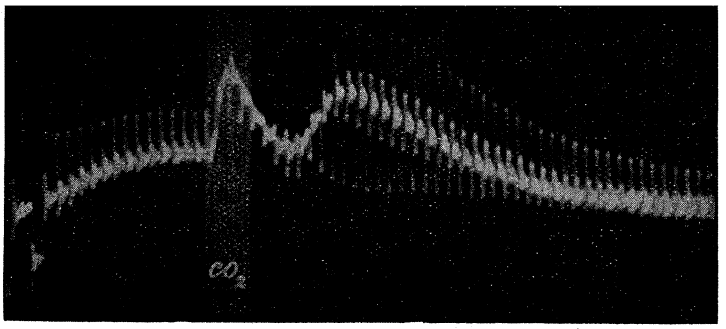
Nerve nine hours after excision, giving a deflection of the third stage, viz., N., temporarily reverted to S. in consequence of tetanisation.

1. Deflection by 0·001 volt.
2. Normal series N.S.
3. Tetanisation for 5 minutes.
4A. Normal series S.N., giving place to
4B. Normal series N.S.

This is one of the few instances in which a negative after-deflection has been observed; the nerve had been employed two hours previously to test upon it the effect of muscle-extract.

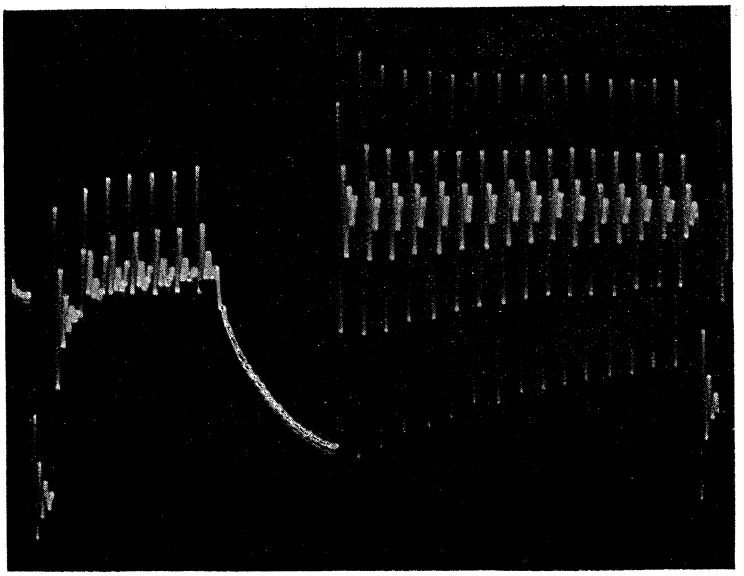
kind) the deflection before 5 minutes' tetanisation is barely visible, the after-deflection before tetanisation is markedly positive ; subsequent to the tetanisation the deflection is markedly negative.

Fig. 41. (843.)



Nerve 11 hours after excision, giving a deflection of the third stage, temporarily reversed, in consequence of CO_2 passed through the nerve-chamber for four minutes.

Fig. 42. (960.)



Nerve 10 hours after excision, giving a variation of the second stage (late) or third stage (early).

At the beginning of the record the deflection is *n.*, and the after-deflection N. With successive excitations the *n.* deflection diminishes and disappears.

With the 5 minutes' tetanus (coil at 10 units) from α to ω, there is no α deflection, a rapidly increasing negativity from α to ω, and a large positive ω deflection. Subsequently the rhythmic excitation produces a large negative deflection gradually decreasing.

Chapter IV.—On Positive and Negative Effects of Excitation.

§ 1. *State of the Question.*

The explanation nearest to hand of positive and negative variations and after-variations caused by excitation, is that which attributes opposite electrical concomitants to the opposite chemical movements of disintegration and reintegration of which all living matter is the seat.

A priori, every movement of disintegration must be made good by a simultaneous or subsequent movement of reintegration, and it was natural to suppose, with Hering and his pupils, that in medullated nerve we have a negative effect significant of the former, followed by a positive after-effect significant of the latter.

In nerve we have hitherto had no evidence of positive effects *during* excitation, other than phenomena that were attributable to the predominance of anelectrotonic currents aroused by the exciting current; at the present time the question of the physiological *versus* physical nature of such currents, their evidently close connection with the true negative variation or current of action, renders the whole problem especially intricate and obscure.*

* Some of this obscurity is due to the varying import and theoretical connotations of the word "electrotonus." The term applies (1) to the permanent extra-polar currents aroused in a nerve that is traversed by a galvanic current; (2) to the similar, but much briefer, extra-polar currents aroused by induction shocks, and summing up to a steady effect when tetanising currents are used; and (3) to the brief propagated change giving rise to a current of action, and summing up to a "negative variation" of a demarcation current.

We may not sharply distinguish between an electrotonic current and a negative variation or action current; the better distinction to make is that between fixed and fugitive electrotonus, fixed electrotonus being a permanent phenomenon pervading the whole length of a nerve in diminishing degree with increasing distance from the polarising electrodes, fugitive electrotonus being a temporary phenomenon propagated along an undisturbed length of nerve in undiminished strength from the point at which a polarising current is suddenly altered. The current of action is a case of such fugitive katelectrotonus (Hermann).

Permanent anelectrotonus (directed in the nerve towards the exciting electrodes) may be aroused by strong tetanising currents of whatever direction, owing to the normal superiority of the anelectrotonic over the katelectrotonic current, and therefore of the anelectrotonic sum over the katelectrotonic sum. Such anelectrotonus, as was pointed out by du Bois-Reymond, in criticism of Moleschott ('Müller's Archiv,' 1861, p. 786, 1862, p. 241), will give rise to a pseudo-positive variation which is in no true antithesis to the negative variation. The latter is due to fugitive katelectrotonus, the former to permanent anelectrotonus.

The positive variation studied in this paper is produced by tetanising currents of minimal strength or little above, and the experimental argument in this chapter is in great measure directed to distinguish this positive variation as not being due to the fallacy of fixed anelectrotonus, but as being in true antithesis to the classical negative variation. To this end anæsthetics were used as described in the text. Anelectrotonus, as a coarse physical fallacy in du Bois-Reymond's sense, is excluded, without prejudice to the nature of the positive variation as a case of fugitive anelectrotonus (= A.), the opposite of the negative variation as a case of fugitive katelectronus (= K.).

Fugitive and permanent electrotonus cannot be sharply distinguished as physiological and physical

Electrotonic currents are clearly the effect of core-polarisation (HERMANN); they are reproducible on polarisable core-models, and in this sense "physical," but they are far better shown on "living" than on "dead" nerve, and in this sense they are physiological. They cannot be sharply distinguished into physical and physiological components. Nor is it possible to draw any sharp line of distinction between a negative variation and an electrotonic current; the former, like the latter, can be reproduced on a core-model (BORUTTAU), and must be regarded as an instance of propagated temporary katelectrotonus.

At the outset of these observations, I sought to distinguish the true negative variation due to a fugitive state of katelectrotonus from the fallacy of the permanent or fixed electrotonic state associated with steady electrolytic polarisation and spread of current. The negative variation provoked by tetanising currents alternating in direction, was found to be independent of that direction, and subject to the influence of anæsthetic and other sedative drugs in an unmistakably physiological manner. This negative variation, subsequently the positive after-variation, and finally the positive variation, were found to be on a similar footing, physiologically modifiable by drugs. The most questionable of these phenomena, the positive variation, which might obviously be due to predominant anelectrotonus caused by alternating currents whatever their direction, was found to be particularly sensitive to the action of reagents, and could therefore not be classed as a fallacy due to exclusively physical polarisation. It must, I think, be admitted to rank as a true action current on the same terms as the negative variation, and, if the latter is propagated katelectrotonus, the former is propagated anelectrotonus. Whether the appearance of one or other of these phenomena is dependent upon the different rate of development of the two states under different conditions, and the consequent predominance of one or other sum of effects under the influence of a given series of induction shocks, is a question into which I cannot now enter. The simple and obvious statement in terms of HERING'S theory (negative variation a sign of dissimilation, positive variation a sign of assimilation) is extremely convenient as a description, but is not finally justified until we are more fully acquainted with the electrolytic changes occurring in the polarisation of living nerve. And although I shall tentatively describe certain facts n terms of that theory, it can only be with the express reservation that the physico-chemical mechanism of anodic and kathodic polarisation may or may not prove to harmonize with such a mode of description. Yet, whether the theory fits the facts or not—whether, *e.g.*, the positive variation be or be not a token of integrative

by means of anæsthetics; they are presumably closely related phenomena of which the electrolytic polarisability of nerve is the common condition, the former requiring a nerve of greater instability than the latter.

In order to escape as far as possible from ambiguity in the use of the terms, the initial letters A. and K. are occasionally (*e.g.*, on p. 64) used to denote fugitive electrotonus, while the full words anelectrotonus and katelectrotonus are used only to designate permanent electrotonus (*e.g.*, on p. 58).

movement, and the anode the pole by which rest, inhibition, and "assimilation" are evoked, in contrast with the kathode evoking action, excitation, and "dissimilation"; or whether, *e.g.*, the conversion of a positive into a negative variation under the influence of carbon dioxide or of prolonged tetanisation be due to altered time-relations of anelectrotonic and katelectrotonic effects—the facts will remain, and doubtless contribute to the future elucidation of this obscure corner in electro-physiology. I think it quite possible that facts which can be most obviously exposed in terms of HERING'S theory of antagonism, may be more accurately described in terms of HERMANN'S theory of electrotonus, yet that in the end the two sets of terms may be found to denote the same opposed chemico-physical changes in living matter.

Meanwhile it is desirable that I should, as briefly as possible, describe the experiments I have so far been able to make in this direction, and the theoretical interpretation of which they appear to be susceptible.

To this end it will be convenient to give (1) a more connected account of experiments already touched upon in Chapter III., concerning the effects of tetanisation; (2) a preliminary account of experiments concerning the action of carbon dioxide on polarisation phenomena; and (3) what seem to be the principal hypothetical inferences based upon these various data.

§ 2. *Effects and After-Effects of "Brief" Tetanisation.*

The ordinary deflection of normal fresh or nearly fresh nerve during tetanic excitation is, as was discovered and determined by DU BOIS-REYMOND, negative to the current of injury. This "negative variation," as was first pointed out by HERING, is frequently followed by a "positive after-variation" (viz., in the same direction as the current of injury) smaller than the preceding negative variation.

In conformity with the plan of connections (fig. 3) observed throughout these observations, this normal galvanometric response *during* and *after* excitation is to be designated by the symbol S.n.

A nerve left to itself in normal saline—(for 1 to 2 hours in summer, for 6 to 12 hours in autumn) will, in all probability, exhibit a response of similar type, in which, however, the positive after-variation is larger than the preceding negative variation, to be designated, therefore, by the symbol s.N.

				Deflection.	After deflection.
1.	Large negative,	followed by	small positive	S.	n.
2.	Small negative	,,	large positive	s.	N.
3.	Nil	,,	positive	0	N.
4.	Positive	,,	positive	N.	N.
5.	Positive	,,	negative	N.	S.
6.	Negative	,,	negative	S.	s.
7.	Nil, or small negative	,,	negative or large negative	0 or s.	S. S.

Later still, especially in summer, or in winter after the nerve has been under prolonged experiment of various sorts, the variation *during*, as well as after, excitation, may be observed to be positive.

It has been convenient to take these three points as being respectively characteristic of three states or stages :—

1st stage, "fresh." Negative deflection larger than positive after deflection, S.n.
2nd stage, "transitional." Negative deflection smaller than positive after deflection, s.N.
3rd stage, "stale." Positive deflection, N.

In the third stage, of which the positive deflection is taken as the characteristic, the after-deflection may be positive (usually) or negative (exceptionally), and, in addition to the responses formulated above, I have observed cases of a negative deflection followed by a negative after-deflection, and of no deflection followed by a positive after-deflection.

The *à priori* conceivable list of possible varieties of response (omitting as of uncertain determination cases of zero after-deflection) is now complete, with one significant exception, which is indicated in parentheses, and has never come under my observation.

Of the above list, the first four members include the types of response that have most frequently come under my observation; they form a sequence, in so far as two or more members may always be observed to follow each other in the order named, from 1 to 4, and may always be made to follow each other backwards from 4 to 1 by the chief disintegration product CO_2 (A) externally applied to the nerve, and (B) presumably evolved from within the nerve itself during a prolonged tetanisation. This reversal of sequence has, in fact, been fully described in Chapter III., where the sequence itself, now to be considered, was unavoidably stated in dogmatic form in preface to a detailed description of its reversal by CO_2. It may, however, be mentioned that a similar reversal is effected by various other means—by formic and lactic acids, *e.g.*, and by a renewed transverse section.

The fifth member of the list, viz., a positive deflection followed by a negative after deflection, N.S., was far less frequently and assuredly observed, and I do not know whether, in order of development, it should be placed before or after the response N.N. The after-effect S. is, in this case, never very pronounced, so that, in view of the uncertainty of instrumental correction considered in the head-note to Table D, it may have been present in slight degree more often than it was obviously present.

The sixth member, viz., persistence of a negative effect as negative after-effect, was also infrequently observed otherwise than as a consequence of CO_2 (when it was practically constant for nerve in the 2nd stage), or of strong stimulation (when it may in part have been a CO_2 effect), or of chemical modification. This infrequency was somewhat surprising in view of the experience of previous observers (HERING

among others), who have noted a depression of current subsequent to the usual negative variation during excitation. I have, indeed, observed such a depression as a regular consequence of strong excitation, but a negative after-effect concentrated enough in time to give an impulse to the mirror of my galvanometers has been comparatively rare. And, as will be shown in connection with the terminal deflection of prolonged tetanisation, negative after-swing is then comparatively rare and only met with in some cases of nerve in the 3rd stage; in the other stages the terminal deflection has always been positive, as well as in most instances of nerve in the 3rd stage under the conditions of my experiments. An immediate negative after-effect of a negative effect must then be exceptional and restricted to certain special conditions of strength and time of tetanisation; prolonged negative after-effect, on the other hand, is a practically constant phenomenon. Positive after-effect following upon positive effect has been as common, as negative after-effect following upon negative effect has been rare.

The seventh member (or pair of members), viz., a negative after-effect larger than a previous negative effect—s.S. or 0S.—has never come under my observation. The opposite case of a positive effect followed by a larger positive after-effect—n.N., or actually 0N.—has been very common.

The possible cases, S.0, N.0, have not been included in the above list for the reason that, though they have both no doubt occurred, the instrumental correction necessary in the case of after-deflection forbids us to conclude to perfect absence of after-effect. Cases occurred, indeed, in which it was calculated as being absent, but in which we may with safety only draw the conclusion "little or none." This is, however, as regards the surveys before us, a comparatively immaterial and purely formal drawback. We may, as the case arises, draw the far more essential conclusion that an after-effect has become more positive or more negative, and plot out a curve of modified after-effects nearly (but not quite) as strictly justified as a curve of modified effects.

The members most essential to our experimental and theoretical study have been the first four, viz., S.n., s.N., 0N., and N.N., which—considering the experimentally somewhat striking case of 0N. as a case of s.N. with vanishing s.—are reduced to three, which are taken as the basis for classifying the states of nerve as first, second, and third. Of these three members the first and third, in which the currents during excitation are respectively negative and positive, are the principal contrasting types. The intermediate type, in which the current after excitation is prominently positive, is preserved as a separate class, partly because it is representative of a very usual condition of experiment, partly because it is the bridge between the two principal extremes.

§ 3. *Effects and After-Effects of "Prolonged" Tetanisation.*

The effects and after-effects hitherto described have been such as are produced by brief tetanisation, lasting 7·5 seconds. We have now to review the effects and after-

effects produced by prolonged tetanisation, lasting 5 minutes, such as was used to test the nerve for CO_2 effects, as described in the previous chapter. And in this review we shall find it convenient to follow the classification made above of three states of nerve—fresh, transitional, and stale.

Fig. 43. (Plate 2148.)

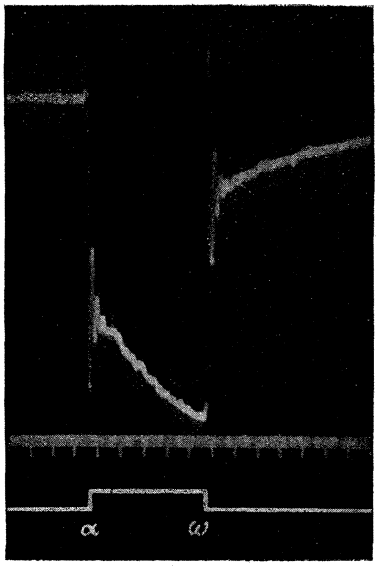


Fig. 44. (Plate 936.)

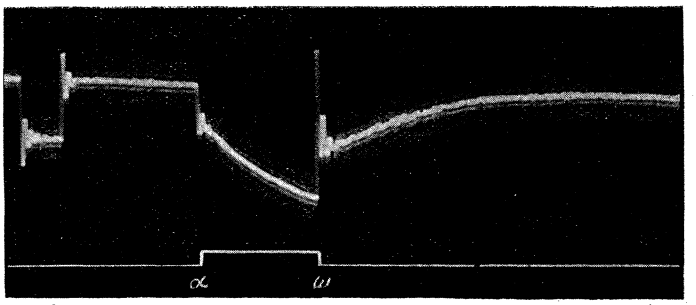
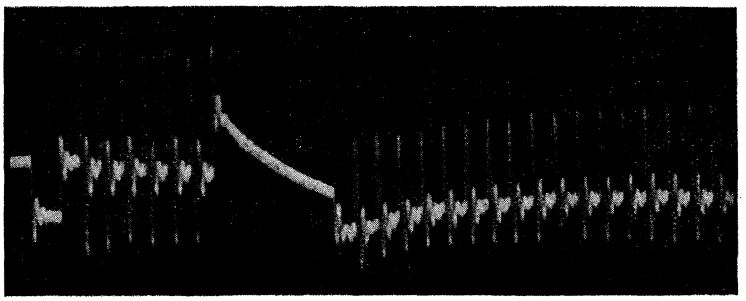


Fig. 43.—February 3, 1896.—Nerve a few minutes after excision, giving a variation of the first stage. Tetanisation for 5 minutes from α to ω; coil at 10 units.

Fig. 44.—December 10, 1895.—Nerve 10 hours after excision, giving a variation of the second stage. Tetanisation for 5 minutes from α to ω; coil at 10 units.

Fig. 45. (874.)



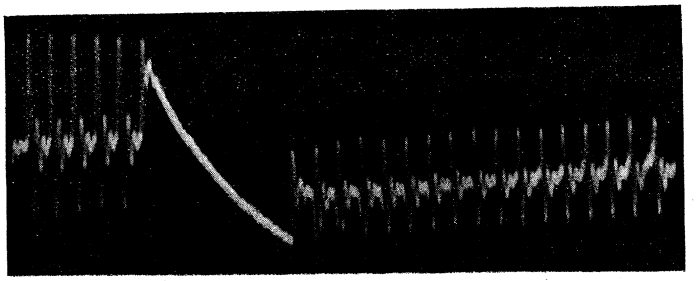
November 30, 1895.—Nerve 10 hours after excision (previously used for observations with ether and with CO_2), giving a variation of the 3rd stage (positive), with a negative ω deflection at the end of 5 minutes' tetanisation, and a diminution of the positive effect of excitation. ("Disguised" effect.)

With nerve in the first and second stages, the alterations of current during and after tetanisation are in accordance with figs. 43 and 44, in which the duration of tetanus is marked from α to ω, α being its initial, ω its terminal, deflection. The α deflection is S. or negative; the course of steady deflection is southward or increasingly negative; the ω deflection is N. or positive; the subsequent state of

current is negative to the state before tetanisation, and gradually becomes less negative.

The curve of increasing negativity during tetanus falls convex to its abscissa, viz., by a diminishing increment, being not far from logarithmic (*e.g.*, figs. 39 and 42); the

Fig. 46. (898.)



December 5, 1895.—Nerve several (?) hours after excision (previously used for observations with sarcosin), giving a variation of the 3rd stage, with a positive ω deflection at the end of 5 minutes' tetanisation, and a reversal of the effect of excitation. ("Clear" effect.)

curve of decreasing negativity after tetanus rises convex to its abscissa by a diminishing decrement (*e.g.*, figs. 44 and 45).

With nerve in the second stage giving the deflection 0N., there is little or no α deflection, and a large positive ω deflection, the current during tetanisation consisting in a gradually increasing negativity (fig. 42).

With nerve in the third state, the α deflection is N. or positive; the course of steady deflection during tetanisation is southwards or diminishingly positive (or increasingly negative); the ω deflection is either negative or positive; the subsequent state of current is negative to the state before tetanisation, and gradually becomes less negative. It was according to the character of the ω deflection that two varieties of third stage effect were distinguished in Chapter III., (a) that in which the ω deflection was N. or positive, and the effect of CO_2 "disguised" (diminution of N.); (b) that in which the ω deflection was S. or negative, and the effect of CO_2 "clear" (replacement of N. by S.) (figs. 45 and 46).

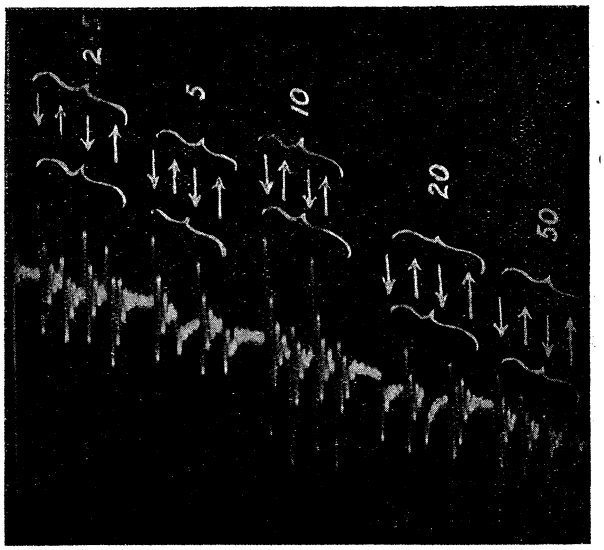
The points of identity and of difference in the tetanic alterations of the three stages may now be summarised as follows:—

State.	Effect before tetanus.	α.	During tetanus.	ω.	Effect after tetanus.
1st	S.	S.	Sward	N.	Increased S.
2nd	s. or 0	s. or 0	Sward	N.	Increased S.
3rd, *a*	N.	N.	Sward	N.	S.
b	N.	N.	Sward	S.	Diminished N.

It will naturally be asked what significance belongs to these facts; what is their dependence, if any, upon the strength and length of excitation in relation to state of nerve, and whether there is any significant relation to be traced between an α and an ω deflection under various conditions, &c., &c. All these questions are difficult to answer; the general question of significance will be considered by itself, other questions of fact, to which owing to the number of variables I can only give incomplete or fragmentary answers, may receive such answer at this stage. But, whereas the more general statements heretofore made are presented as generalizations resting upon a number of particular experiments considered as affording a broad enough basis, the following are to be regarded almost in the light of particular observations, which a more extended survey may (or may not) demonstrate to be representative of general relations. Separate investigations of considerable extent will be required before this conclusion may safely be arrived at.

Strength of Excitation.—I have observed more than once (but am unable to reproduce the experiment at will) that nerve in the 3rd stage giving with weak tetanisation the deflection N., gives with stronger tetanisation the deflection S. The following is a case in point (826), which is reproduced in fig. 47, partly in order to

Fig. 47. (826.)



give an example of how such measurements are made, partly because a critic may see in it internal evidence of some possible explanation or fallacy that I am unable to detect. The facts here signify possibly that the nerve has been in such a state that fixed anelectrotonus was manifested at lower current strength than fugitive katelectrotonus. This possibility of a fixed anelectrotonic component is further supported

by Obs. 2149,* where, with constant strength but diminishing interpolar distance, the negative variation was diminished, viz., at 15 millims. it was — 17, at 10 millims. it was — 8, at 5 millims. it was 0.

Strength.	Deflection (descending break).	Deflection (ascending break).
2·5 units	N. 9·5	N. 3·5
	N. 9	N. 7
5 ,,	N. 9	S. 5
	N. 8·5	S. 6·5
10 ,,	N. 13·5	S. 14·5
	N. 13	S. 12
20 ,,	S. 9	S. 18
	S. 7	S. 18
50 ,,	S. off plate	S. off plate
	S. off plate	S. off plate

The galvanographic curve of a prolonged tetanic variation is extremely like that of an ordinary muscular tetanus, both as regards the gradual tetanic increase and the

* December 1, 1896.—Obs. 2149 was somewhat exceptional; other observations, of a presumably more typical character, made for the purpose of comparing the effects of tetanisation with assuredly electrotonic effects, showed no difference of the negative variation with different distances between exciting and leading-off electrodes, *e.g.*:—

Obs. 2107.—Leading-in electrodes, 1 centim. apart. Leading-out electrodes, ditto.

Length of nerve between leading-in and leading-out electrodes.	Anelectrotonic current by 1·4 volt.	Katelectrotonic current by 1·4 volt.	Negative variation by tetanisation at 15 units, in both directions; 66 interruptions per sec. for 7·5 secs.	
centims.				
3	+ 1·5	— 0·5	—3·5	—4·0
2	+ 7·5	— 2·5	—4·0	—4·0
1	+120·0	—90·0	—4·0	—4·5

December 5, 1896.—Exp. 2131. Nerve giving a deflection of the third stage. Intrapolar distances = 1 centim. each; polarising current by 1 Leclanché; 0·001 volt gives 13 millims. deflection.

Length of nerve between leading-in and leading-out electrodes.	Anelectrotonic current.	Katelectrotonic current.	Positive variation by tetanisation at 15 units, in both directions for 7·5 secs.; 66 interruptions per sec.	
centims.	millims.			
3	+ 0·5	Trace	+8·5	+8·0
2	+ 2·0	— 1·0	+8·0	+8·0
1	+34·0	—23·0	+8·0	+8·0

gradually diminishing after-tetanic remainder. In nerve, at least, the effect is attributable to CO_2 gradually evolved during the tetanisation, gradually dissipating itself (or, perhaps, "reinvolved") after tetanisation. Owing to the gradual increase of the negative deflection during tetanus, it always happens in the first, and still more markedly in the second state of nerve, that the ω positive swing is greater than the α negative swing. (I did not, of course, neglect to verify the equality of the two opposed swings caused at make and break of a prolonged constant current; proof of this equality is, indeed, afforded by the standard deflection of $\frac{1}{1000}$ volt, taken at the beginning and end of most of the observations, *e.g.*, the standard deflection at the end of fig. 42.) With nerve in the 3rd stage, in which the α deflection is positive, the ω swing may be greater, equal to, or smaller than the α swing; it may be positive, negative, or so small as to be of doubtful direction. It is negative and smaller than the α deflection if, during tetanisation, the positive state has only diminished. It is positive and either smaller or larger than the α deflection if, during tetanisation, the positive has given place to a negative state. A negative ω deflection smaller than the corresponding positive α deflection is present in figs. 32 and 45; a positive ω deflection smaller than the corresponding positive α deflection is present in fig. 46; a positive ω deflection larger than the corresponding positive α deflection is present in figs. 31 and 46.

There is, as might be expected, a fairly regular relation between the difference of the α and ω swings and the deflections of the minute before and minute after tetanus. This relation is most regular in the 1st and 2nd stages, where the two differences are greater and smaller together.

	$\omega-\alpha$.		d_2-d_1.*
E.g., in 741 (fig. 29) the two differences are respectively . .	15	and	7
,, in 689 the two differences are respectively	18	,,	8
,, in 761 ,, ,, ,, ...	7	,,	4
,, in 876 ,, ,, ,, ...	4·5	,,	2·5
,, in 960 (fig. 42) the two differences are respectively . .	45·5	,,	34

Other instances are contained in Table D.

The relation is not so clear nor so constant in the third stage, where the α deflection is + and the ω deflection + or −. With a positive ω it generally happens that the larger the ω the larger the succeeding negative deflection, *i.e.*, the greater the difference between that deflection and the deflection immediately preceding the tetanus.

E.g., in 933 (fig. 40) $d_1 = +\ 6{\cdot}5$, $\alpha = +\ 6{\cdot}5$, $\omega = +\ 30$, $d_2 = -\ 15{\cdot}5$,
,, in 898 (fig. 46) $d_1 = +\ 13$, $\alpha = +\ 13$, $\omega = +\ 12$, $d_2 = -\ 6$.

* d_1 signifies the last ordinary deflection preceding tetanisation, d_2 the first ordinary deflection subsequent to tetanisation.

With a negative ω, it generally happens that with larger and smaller ω are associated smaller and larger diminution of an original deflection.

E.g., in 874 (fig. 45) $d_1 = +17$, $\alpha = +17$, $\omega = -7{\cdot}5$, $d_2 = +12$,
„ in 782 $d_1 = +11$, $\alpha = +11$, $\omega = -11$, $d_2 = +11$,
„ in 942 $d_1 = +11$, $\alpha = +11$, $\omega = -23{\cdot}5$, $d_2 = +13{\cdot}5$.

These last three examples are chosen to illustrate the, for the moment, empirical relation between the two differences, and include what have been referred to as exceptional effects of tetanus (viz., augmentation of a north, diminution of a south deflection). As may be noticed in the three cases, with a negative ω smaller, equal to, and larger than the corresponding positive α, we have a positive d_2 smaller, equal to, and larger than the corresponding positive d_1.

§ 4. *Preliminary Account of the Action of* CO_2 *upon Polarising and Electrotonic Currents. Polarisation Increments and Electrotonic Decrements.*

Experiments under this head were undertaken principally with the object of determining whether or no the positive variation upon which I had studied the action of reagents was a phenomenon of ordinary fixed anelectrotonus or a fugitive effect, the counterpart of the negative variation. They were commenced on the inadequate supposition that ordinary electrotonic effects, being reproducible on the core-model, are physical phenomena independent of the physiological state of nerve, and should therefore be refractory to the action of anæsthetics. On this supposition anæsthetics should have furnished the distinction between electrotonic (physical) and excitatory (physiological) effects, and the positive variation would at once have been placed in one or other category.

The effects of anæsthetics (carbon dioxide, ether, chloroform) were studied upon:—

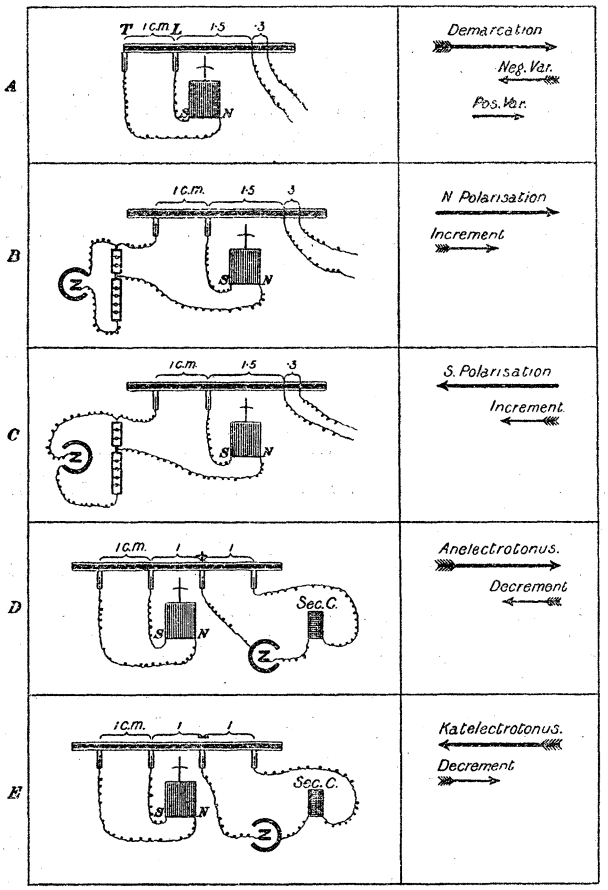
1. Polarising currents; 2. the excitatory increments of polarising current; 3. electrotonic currents; 4. the excitatory decrements of electrotonic currents, and although the simple distinction just alluded to could not be absolutely established, certain results were obtained calculated to further our comprehension of electrotonic effects and indirectly to establish the positive variation as a physiological phenomenon on the same footing as the negative variation.

Detailed description of these experiments will be given on a future occasion; at this juncture only the briefest possible summary of results is presented, in so far as these are connected with the influence of carbon dioxide upon the polarisation phenomena of which the electrical effects of excitation are a special case.* The

* Boruttau's recent demonstration (Pflüger's 'Archiv,' vol. 58, p. 1, 1894, and vol. 63, p. 145, 1896), on core-models of platinum in saline solution, of a wave of negativity provoked by tetanisation, travelling with measurable velocity (150 to 170 metres per second) and not very marked decrement, thus giving rise to a typical diphasic variation, has very completely fulfilled the identification of the electrical phenomena in excited nerve with polarisation phenomena, in accordance with Hermann's point of view.

diagram (fig. 48) enumerating the several currents that have been tested by anæsthetics, and the eight plates (figs. 49 to 56) giving typical results of the action of carbon dioxide, will most clearly and briefly fulfil this purpose.

Fig. 48



Plan of the several currents referred to in the text and recorded in the next group of 8 plates.

The connections are such that in each case the direction of the arrow indicates the direction of current in the nerve and in the galvanometer.

Current in the nerve from left to right is indicated by current in the galvanometer from S. to N. ("positive"), reading upwards on the final record; and *vice versâ*.

For electrotonic currents and their excitatory decrements the polarising and exciting circuits were conjoined, and completed through the nerve by the same pair of unpolarisable electrodes.

Anæsthetics (represented in the figures by CO_2)* produce no effect upon the strength of polarising currents, but, as was to be expected, polarisation increments and electrotonic decrements, which are essentially action-currents, are modified by

* Instances of the action of Et_2O and of $CHCl_3$ are given in a paper dealing more especially with the question of anæsthetics in current numbers of 'Brain' (1896). Some experiments are quoted in Table E, and the subject will be dealt with more fully in a future paper. As regards carbon dioxide the influence of quantity, and the effects of tetanisation will be there considered.

CO_2, and ordinary electrotonic currents, contrary to expectation, although apparently less sensitive, are distinctly altered.

We cannot, therefore, sharply distinguish between an ordinary electrotonic and an action-current by means of anæsthetics, even though we may have found that the latter is more easily affected than the former. Electrotonus is a physical phenomenon inasmuch as it occurs on any polarisable core model, and, in such case, it is not affected by anæsthetics. But it is also a physiological phenomenon, inasmuch as the chemical instability, viz., polarisability, characteristic of the living nerve (and no doubt in less degree retained by nerve that is called "dead") is essential to its manifestation. If the nerve is temporarily fixed by an anæsthetic, or permanently fixed by a poison, *e.g.*, by aconitine, the polarisability and depolarisability that are essential to the manifestation of electrotonic phenomena no longer exist, and the nerve loses, first its capability of undergoing the transmitted and temporary effect ("fugitive electrotonus"), next its capability of undergoing the permanent effect ("fixed electrotonus").

This conclusion seems to be not entirely in harmony with BIEDERMANN'S results,* partly, perhaps, by reason of different senses attaching to the term electrotonus, partly, perhaps, by reason of differences in the conditions of experiment.

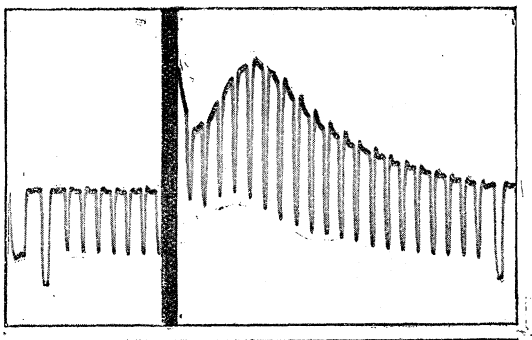
BIEDERMANN distinguishes in the extra-polar current produced during and after the passage of a constant current (1 to 3 volts, 10 millims. between polarising and galvanometer electrodes) a physiological factor dependent on propagated excitation in the living nerve and a physical factor dependent upon its anatomical integrity. The former "physiological electrotonus") he finds to be subject to *temporary* suppression by "brief" (5 to 10 minutes) etherisation. The latter ("physical electrotonus") he finds to be relatively more stable, but to undergo diminution and *definitive* abolition by further "prolonged" etherisation destructive of the nerve. Current that is temporarily suppressed by etherisation is regarded by BIEDERMANN as an expression of physiological electrotonus; the residual current, that can only be suppressed, and then finally so, by "prolonged" etherisation, he considers as being due to physical electrotonus.

I find that electrotonic as well as action currents (otherwise expressed permanent as well as fugitive electrotonus) may be *temporarily* suppressed by anæsthetics. And whereas BIEDERMANN from his observations adopts anæsthetics as distinguishing between physiological and physical electrotonus, I from mine do not feel entitled to make any sharp distinction by such means between the ordinary electrotonic and the true action current. Both have been sensitive although unequally so, and, much as I desired to draw the line between electrotonus and action-current, I did not feel it safe to do so by means of anæsthetics, which in my hands have served rather to draw the line between electrotonus and current diffusion.† The latter is especially liable to

* 'Electrophysiologie,' vol. 2, p. 695, Jena, 1895.

† I have Professor HERING'S authority for stating that the term "physical electrotonus" arose in his laboratory as an expression denoting neither more nor less than "current-diffusion." (Leipzig, Dec. 22, 1896.)

Fig. 49.



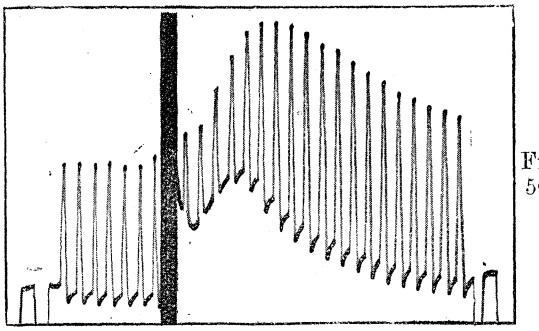
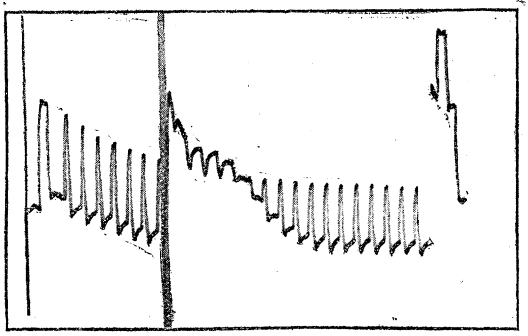


Fig. 50.

Fig. 51.



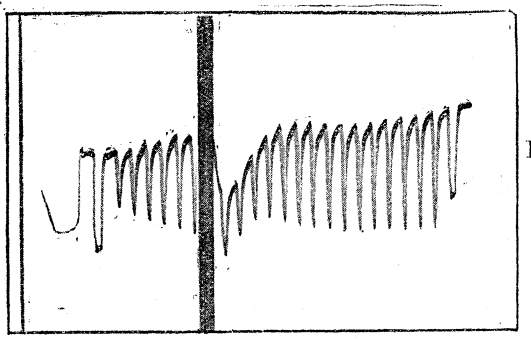
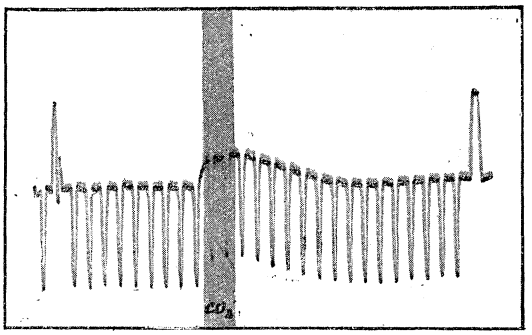


Fig. 52.

Fig. 53.



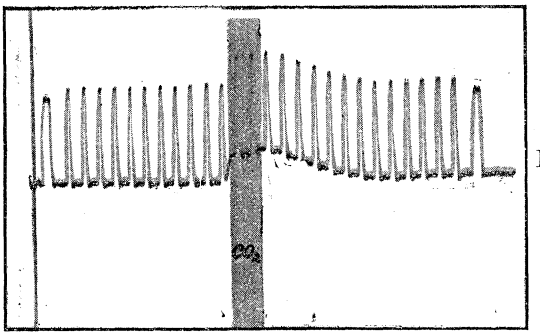
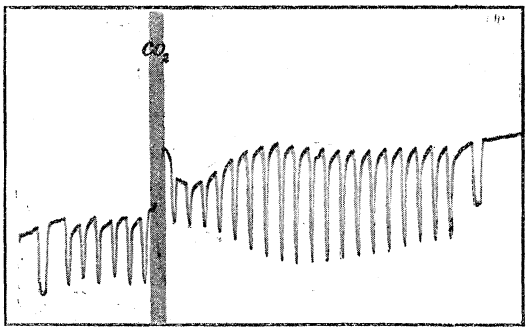


Fig. 54.

Fig. 55.



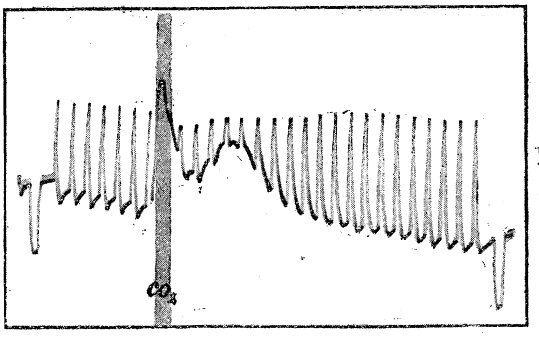


Fig. 56.

Fig.	Plate.		Fig.	Plate.	
49	2195	Effect of CO_2 on katelectrotonus	50	2199	Effect of CO_2 on anelectrotonus
51	2181	„ „ a katelectrotonic decrement	52	2185	„ „ an anelectrotonic decrement
53	2207	„ „ a S. polarising current	54	2208	„ „ a N. polarising current
55	2204	„ „ a S. polarisation increment	56	2203	„ „ a N. polarisation increment

The above eight records were taken by the dead-beat galvanometer, with connections as indicated in fig. 48.

In the observations on katelectrotonus and anelectrotonus (figs. 49 and 50), the polarising current was

by one Leclanché cell (= 1·4 volt) with distances (*a*) between polarising electrodes, (*b*) between polarising and galvanometer electrodes, (*c*) between galvanometer electrodes, each = 10 millims. An interrupter (make = 15 secs., interval = 45 secs.), was placed in the polarising circuit.

In the observations on the two electrotonic decrements (figs. 51 and 52), the polarising and exciting currents entered the nerve by the same pair of unpolarisable electrodes (figs. 48D and 48E), the interrupter was in the exciting circuit.

In observations on the polarisation increment (figs. 55 and 56), the polarising current was taken = 0·05 volt. (figs. 48B and 48C). The interrupter was in the exciting circuit.

occur in such experiments, where a comparatively high electromotive force (over 1 volt) is turned on at intervals. When, in consequence of an anæsthetic or toxic drug, an originally unequal anelectrotonus and katelectrotonus has given place to a reduced effect equal in the two opposite directions, the latter has in some instances disappeared after the nerve has been completely crushed in the interpolar region, while in other instances it has persisted little if at all diminished, *i.e.*, in some instances (Table E, Obs.) the residual deflection has been attributable to BIEDERMANN'S "physical electrotonus," in others it has clearly been due to current escape. But the point is one requiring further study; as regards the present investigation, it is a side-issue.

With regard to a positive variation, it might possibly be due to—

(1). An ordinary anelectrotonic current.
(2). A negative variation of an ordinary katelectrotonic current.
(3). A north polarisation decrement.
(4). A fugitive anelectrotonic current.
(5). Current escape.

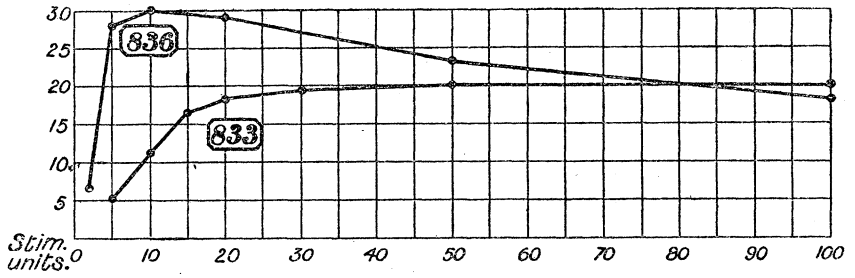
The possibilities (2) and (3) may be dismissed at once; the positive variation cannot be a negative variation of a katelectrotonic current in the absence of katelectrotonus, nor a polarisation increment in the absence of a polarising current. And the fifth alternative, which certainly might, and does appear with an imperfectly insulating moist chamber, besides being excluded by the usual test of crushing in the interpolar region, is further dismissed by the temporary action of CO_2 and other anæsthetics. We are thus left in presence of the alternatives (1) and (4).

The positive variation is produced by very weak currents, and *sometimes* is lost when the current is strengthened. It is extraordinarily sensitive to reagents, especially to a presumably infinitesimal amount of carbon dioxide which leads to its diminution or reversal. Like the negative variation, but unlike an ordinary anelectrotonic current, it does not depend upon the distance between the leading-in and the leading-out electrodes. Confirmatory evidence is, I think, afforded by the absolute identity in the effects of carbon dioxide (and other reagents) upon a positive after-deflection and a positive deflection. The former (HERING'S positive 'Nachschwankung'), is a state of predominantly positive reaction consequent upon a

previous state of predominantly negative action, interpretable as a physiologico-chemical assimilation consequent upon previous dissimilation. Or, in electrotonic language, it is an anti-katelectrotonic (fugitive) after-effect. If so, and if the positive after-deflection is of the same genus as the positive deflection, then the interpretation good for the former is good also for the latter.

In the case of the negative effect, the range from minimal to maximal is extensive, and the maximal effect is, as a rule, gradually and indefinitely increasing. In that of the positive variation, the range from minimal to maximal is narrow; in general rule a positive maximum has been reached with a strength of tetanisation not greatly in excess of the minimal effective, and has undergone more or less pronounced diminution with further increased strength of tetanisation; in a few instances (*e.g.*, fig. 47), with such increase, a positive has given place to a negative deflection. If the positive variation were due to ordinary anelectrotonus, these relations should be reversed; the positive variation should increase indefinitely, and a negative variation at lower stimulation-strength should have given place to a positive variation at higher stimulation-strength. This I have never witnessed.

Fig. 57.



	Strength of stimulation.	Magnitude of deflection.	Value of 0·001 volt.	
833	units.	millims.	millims.	Break desc.
	5	− 5·5	48	
	10	− 11		
	15	− 16·5		
	20	− 18		
	30	− 19·5		
	50	− 20		
	100	− 20		
836	1	+ 0	22	Break desc.
	2	+ 6·5		
	5	+ 28		
	10	+ 30		
	20	+ 29		
	50	+ 23		
	100	+ 18	22	

Observations 833 and 836 (figs. 58, 59), illustrate this point, especially if their respective measurements are plotted out as curves (fig. 57). The former gives the series of increasing negative variations of a nerve of the 1st state, the latter, the series of diminishing positive variations of a nerve of the third state.

The positive after-effect of the second state of nerve undergoes a similar decrease with increasing strength of stimulation, as may be anticipated from its regular decrease, with series of brief tetanisations of constant strength. Whereas, as previously stated, the negative effect of such nerve undergoes staircase increase, its positive effect undergoes staircase decrease, phenomena that are attributable to an

Figs. 58, 59 (Plates 833, 836).

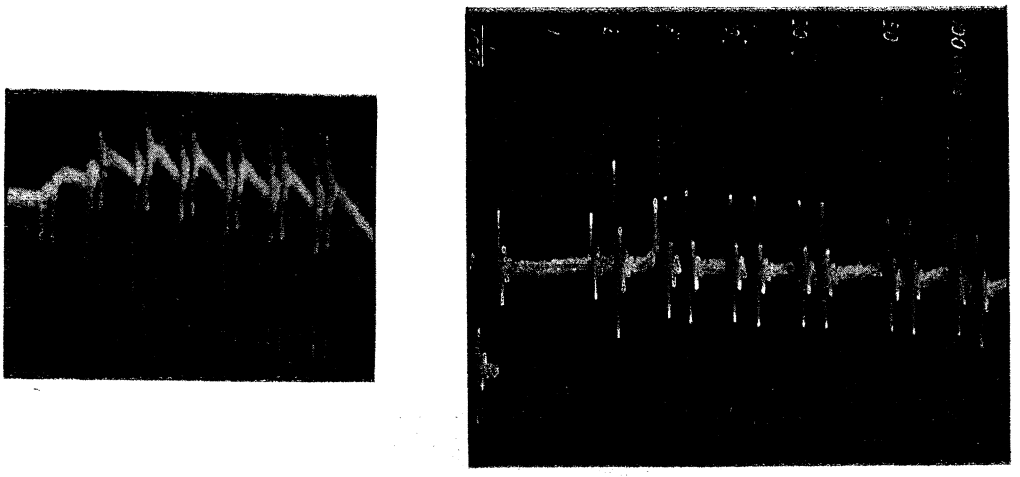


Fig. 58.—Records from which the measurements were taken for the construction of the curves of fig. 57.
Fig. 59.—Excitations in pairs, first with break descending, *i.e.*, towards the galvanometer electrodes, then with break ascending. Only the former have been used for the construction of the curves.

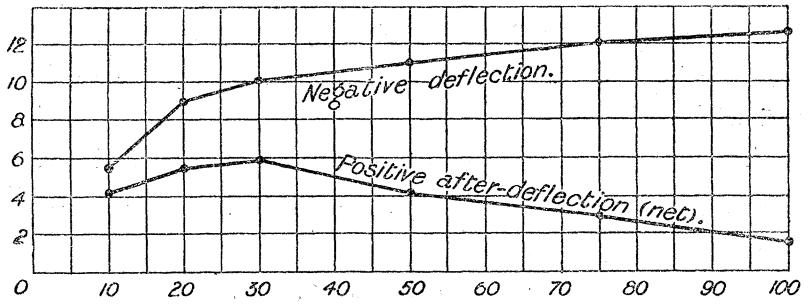
evolution of carbon dioxide, and the second of which further bears out the identity in kind between the positive effect and the positive after-effect. This point, incidentally present in several other figures of this paper, *e.g.*, in fig. 66, is more particularly illustrated by the plotted curves of fig. 60, with which are given the actual records from which this figure is derived (figs. 61 and 62).

Finally, we may comment upon a point already mentioned at an earlier stage, viz., the rarity of a negative as compared with a positive after-effect; it has most commonly happened that a positive after-deflection has taken place succeeding not merely a negative but also a positive deflection; if the positive deflection had been due to ordinary anelectrotonus it should have been followed by a negative after-deflection.

The weight of evidence is thus against the ordinary anelectrotonic nature of the positive variation produced during tetanisation, and towards its recognition as an

excitatory phenomenon, similar to the positive after-variation, and the counterpart of the negative variation.

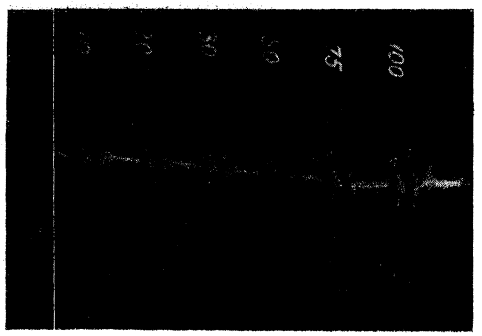
Fig. 60.



2070

Strength of stimulation.	Magnitude of negative deflection.	Magnitude of positive after-deflection.*
10	− 5·5	+4·3
20	− 9	+5·5
30	−10	+5·8
50	−11	+4·2
75	−12	+3·0
100	−12·5	+1·7

Fig. 61. 2070.



Record from which the measurements were taken for the construction of the curves in preceding figure. Excitation in pairs, first with descending break, then with ascending break, only the former being used for the curves.

§ 5. *Theoretical Considerations.*

In conclusion, but explicitly recognizing that the conclusion may be otherwise stated in the particular terminology of electrotonic phenomena, we may admit as a general theory, embracing the positive and negative expressions of action in living nerve, excited by electrical stimulation, that :

* Corrected as described on p. 66, note to Table D.

Neuroplasm is chemically mobile in two directions, downwards in the direction of disintegration, upwards in the direction of integration.*

Although we are often constrained to refer to these two opposite aspects of metabolism as two opposite processes—a negative process, disintegration, dissimilation, analysis, katabolism as opposed to a positive process, integration, assimilation, synthesis, anabolism—they should not be imagined as distinct, but as conjoined in one actually indivisible complex change. We may not suppose that we ever witness the simple effect of a positive or of a negative process acting alone, but always the resultant effect of a process in which negative predominates over positive, or *vice versâ*. The process behind its negative or its positive manifestation is not exclusively negative or exclusively positive, but negative (and positive), or positive (and negative).†

In terms of this hypothesis or figment, the phenomena previously described relating to the three states of nerve, may be expressed as follows :—

1. In fresh nerve ("first state"), containing neuroplasm highly wound up, *i.e.*, capable of unwinding, excitation, which augments the negative-cum-positive process, elicits a manifest negative resultant, signified galvanometrically by a negative deflection. During the period subsequent to predominant disintegration, reintegration is predominant, but since the conjoint positive-cum-negative process is taking place in neuroplasm that is still at a high level, that positive predominance is slight and the positive after-effect is small.

2. In stale nerve ("third state"), containing neuroplasm that has run down, *i.e.*, that is capable of being wound up, excitation, which augments the negative-cum-positive process, elicits a manifest positive resultant, and there is, *even during excitation*, a predominant integration, signified galvanometrically by a positive deflection.

This predominance of positive over negative diminishes during tetanisation (? by reason of CO_2), and may give way to a predominance of negative over positive. So long as there is positive predominance during excitation, the deflection (ω), at its close, will be negative (fig. 45); when negative predominance has set in during excitation, the deflection (ω), at its close, will be positive (fig. 46). (In the former case, the "disguised" effect of CO_2 is witnessed, in the latter, the "clear" effect.)

3. In "transitional" nerve ("second state") containing neuroplasm that is half run down, capable therefore of being wound up or unwound, the negative factor predominates during excitation, and the positive factor predominates after excitation.

* Using the term "neuroplasm" to denote the living excitable matter that forms the axis-cylinder of a medullated fibre, in distinction from the sheath itself which, in relation to the medulla, is the "trophoplasm."

† The ordinary "negative variation" of nerve, due to predominant negative change at and near the longitudinal lead-off, is itself probably (if not demonstrably) composed of two factors—negative predominant over positive near the longitudinal lead-off, and positive predominant over negative near the transverse lead-off.

The disposition to movement in one or other direction is more nearly equipoised than in either the first or the third states.

4. In all states of nerve the negative change is promoted by little CO_2, obstructed by much CO_2.

5. In all states of nerve the usual effect of prolonged tetanisation is an augmentation of the negative mobility (augmented S in the 1st and 2nd states, figs. 29, 35; diminished N and reversal from N to S in the 3rd state, figs. 30, 32); the exceptional effect of tetanisation is a diminution of the negative mobility (diminished S in the 2nd state, fig. 37; increased N in the 3rd state, fig. 36).

Expressed in terms of electrotonic changes K and A, the same series of facts may be clothed in the following statements, involving less assumption, but also offering less explanation:—

1. In fresh nerve (1st state) the sum of propagated katelectrotonic effects exceeds the sum of propagated anelectrotonic effects—$K > A$. Therefore, negative deflection during tetanisation.

2. With lapse of time, the difference $K > A$ diminishes to $K = A$, and becomes reversed to $K < A$. Therefore, positive deflection during tetanisation.

3. The effect of CO_2 in small quantity is to augment K. Therefore, augmented negative deflection of the 1st and 2nd states; diminution or reversal of the positive deflection of the 3rd state.

4. The effect of CO_2 in large quantity is to diminish K. Therefore, diminution or abolition of negative deflection of 1st and 2nd states.

5. The usual effect of tetanisation is augmented K; the exceptional effect of tetanization is diminished K.

6. During prolonged tetanisation the difference $K > A$ of the 1st and 2nd states gradually increases, and the difference $A > K$ of the 3rd state gradually diminishes. Therefore, the deflection during such tetanization is increasingly negative or diminishingly positive.

7. If during prolonged tetanisation of nerve in the 3rd state, the diminishing difference $A > K$ reaches to reversal, viz., to $K > A$, the deflection at the end of tetanisation is positive; fig. 46. If the difference $A > K$ does not reach to equality, $A = K$, the deflection at the end of tetanisation is negative; fig. 45.

§ 6. *Concerning the Unfatiguability of Nerve.*

The experimental inexhaustibility of nerve-fibres as compared with the rapid exhaustibility of nerve-terminals, upon which, in common with Wedenski and Bowditch, I have formerly laid much stress, and which, as mentioned in Chapter I. (p. 2), formed one of the starting-points of this investigation, presents itself with a very different and far more readily intelligible significance by the light of these considerations, fortified by what I am tempted to designate as proof of a chemical

alteration in tetanised nerve. And in this connection it is of little moment whether we hold, with HERING, that negative effects and positive after-effects are the signs of dissimilation and assimilation, or with HERMANN, that they are of electrotonic nature —the signs of the extraordinarily rapid polarisation and depolarisation characteristic of living medullated nerve.* Medullated nerve-fibres are experimentally inexhaustible, not because their tetanisation effects little or no chemical change, but because such change is extremely rapid and rapidly effaced by a change in an opposite direction. The materials of which normal medullated nerve is composed (grey "neuroplasm" and white "trophoplasm") are in a state of great chemical lability, so that whatever is done, is done easily, and as easily undone.† That something is done (or rather undone) during nerve-tetanisation, has, I think, been proved by the series of experiments described in Chapter III.; and towards the determination of what that something may be, I have also brought evidence, if not proof, that one at least of its terminal products is carbon dioxide.

* HERMANN. 'Polarisation der Muskeln und Nerven.' PFLÜGER'S 'Archiv,' vol. 42, 1888.

† At the last Physiological Congress (Berne, 1895) records were exhibited of the retinal currents of action provoked by illumination at regular intervals, and demonstrating that the retina resembles nerve with respect to its inexhaustibility. The interpretation given above for nerve—viz., apparent inexhaustibility as being in reality rapid disintegration effaced by rapid reintegration—applies with even greater probability to retinal phenomena, where, chiefly by reason of HERING'S arguments, we have already been familiarized with the dual aspect of metabolism. The fact that the retina exhibits no sign of fatigue is indeed, in some degree, a collateral item of evidence in the direction of the conclusion *re* nerve. It may scarcely be doubted that the retina excited by light suffers chemical change, and it may be demonstrated that the retina submitted to a practically interminable series of flashes exhibits no signs of expenditure of matter, the strict conclusion is, therefore, that disintegration is effaced by reintegration.

Fig. 62. (Plate 51.)

February 5, 1895.—Portion of a prolonged series of retinal deflections caused by stimulation of the frog's eyeball at intervals of one minute. Each stimulation consists in the light of a standard candle at a distance of two feet acting for a period af $7\frac{1}{2}$ seconds. The electromotive value of each deflection is about 0·0001 volt.

TABLE D.—Measurements of some of the Observations given in Tables B and C.

NOTE.—In most experiments a partially damped Thomson galvanometer (R = 15,000 ohms.) was used, shunted if necessary. In some of the experiments the position of the controlling magnet was such that the suspended system of magnets oscillated between 4 and 5 times per minute (period = 12 to 15 secs., decrement = 2·8 to 3). In others the oscillation frequency was 7 per minute (period = 8·5 secs., decrement = 2).

In any evaluation of after-deflection this purely instrumental oscillation must be reckoned with, and to this end it is best to take the relation between swing and swing-back by letting a small E.M.F. into the circuit for $7\frac{1}{2}$ seconds (*i.e.*, through the mercury pool to be used in the exciting circuit). The corresponding correction of after-deflection is made throughout the numbers given in Table D.

In later experiments I employed a less sensitive, but perfectly dead-beat galvanometer (by Messrs. MUIRHEAD & Co.), the rising or falling time of which was 12 seconds. With the partially damped galvanometer any positive or negative after-effect obtaining during the first few (2 to 7) seconds after the end of excitation was manifested as an augmentation or diminution of the instrumental swing-back; with the aperiodic instrument any such after-effects occurring during the falling time (12 seconds) were lost to view, and only such as remained present after the expiration of that time were manifested. Plate 2175 exhibits the photogram of the dead-beat instrument acted upon by $\frac{1}{1000}$ volt for 1 minute. Plate 2177 is an example of a normal tracing of nerve in a late first or early second stage, the duration of tetanus at 10 units being 15 seconds. It exhibits the gradual decline of a positive after-effect, such as was observed by HERING, and which in my view is a gradually increasing effect of CO_2. Plates 2181, 2185, 2195, 2200, 2203, 2204, 2207, 2208 were likewise recorded with the dead-beat gavanometer. All others were taken with the same partially damped instrument.

The capillary electrometer is in many respects the best instrument of all for obtaining a dead-beat record of the entire effect and after-effect of tetanisation. For various reasons that need not be detailed, I have not yet employed this instrument systematically, but only occasionally, and shall, therefore, not further consider it at this juncture.

The numbers indicate millimeters of deflection (Column I.) and of after-deflection (Column II.). The latter series of numbers requires to be corrected for oscillation, as stated in text. To this end, in observations after No. 675, a key-plate is taken by sending a small E.M.F. into circuit once a minute through the mercury pool (7·5 seconds), to be used in the exciting circuit.

Plate 763 (7 oscillations per minute) is the key-plate to Nos. 675, 676, 678, 680, 689, 710, 711, 737 741, 745, 747, 751, 752, 761, 762, 782.

Plate 2041 (between 4 and 5 oscillations per minute, more rapidly moving surface) is the key-plate to Nos. 825, 843, 857, 858, 859, 869, 874, 898, 905, 912, 928, 929, 933, 935, 937, 942, 958, 960, 982, 983, 984, 985, 990, 991, 993, 994, 2003, 2004, 2005, 2006, 2054, 2055, 2057, 2068, 2069, 2072, 2073, 2074, 2075, 2080, 2081, 2082, 2084.

Correction is accordingly made by subtracting from the after-deflection (Column II.) either one-third or two-thirds of the corresponding deflection in Column I., thus yielding a net value in estimation of after-effect due to the nerve.

Great accuracy may not be claimed for such an estimate; the key-plate 763 shows considerable irregularity of swing-back, which, however, was much diminished in subsequent experiments. The net values, after subtraction of one-third deflection, can, therefore, only be approximately correct, and none but well-marked alterations of such values may be admitted to rank as assured data. Key-plate 2041 is more regular, but in the group of experiments to which it belongs the controlling magnet was slipped slightly up and down during adjustment; the oscillation frequency was thus varied between a little more and a little less than 4·5 oscillations per minute, and a relation between swing-back and swing that was ·6 at the higher and ·66 at the lower frequency.

These considerations apply only to the after-deflection, not to the deflection, and are entered upon to show within what range of error the former may be estimated. Looking back to records taken when no heed was paid to the after-effect, it is still possible to form some judgment as to its presence and amount. Instances of such retrospective estimates are given at the end of Table D, referring to some of the plates figured in the paper, viz., Nos. 200, 309, 334, 537, 708; the two pairs of records here figured, 3180–1, 3188–9, with their measurements, are given to show how the oscillating and dead-beat instruments respond to the same electromotive change, and to what extent the corrected measurements of the oscillating instrument are to be trusted.

With the oscillating magnet (5 osc. per minute) the net after-deflection is due to the state of nerve during the 2 to 3 seconds succeeding the end of excitation; with the fully damped magnet (falling time = 12 seconds), any after-deflection is due to the state of nerve during the period commencing 12 seconds after the end of excitation. So that the indications of the two instruments are complementary, the first being that of the beginning and the second that of the tail of the after-effect in the nerve. The two indications have always been of the same sign, the former being the more sensitive of the two. It was frequently noticed that the same nerve giving a considerable net after-deflection to the oscillating magnet, manifested no sensible after-deflection to the damped instrument.

Fig. 63. Key-plate 763.

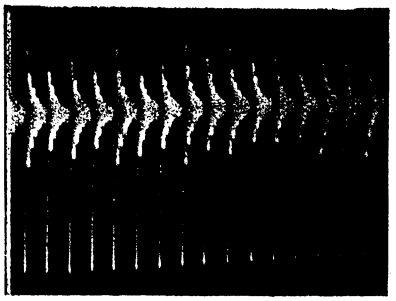


Fig 64. Key-plate 2041.

Fig. 65.

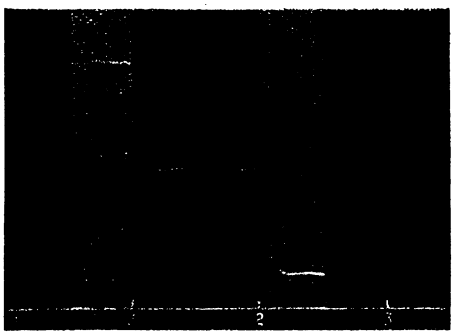


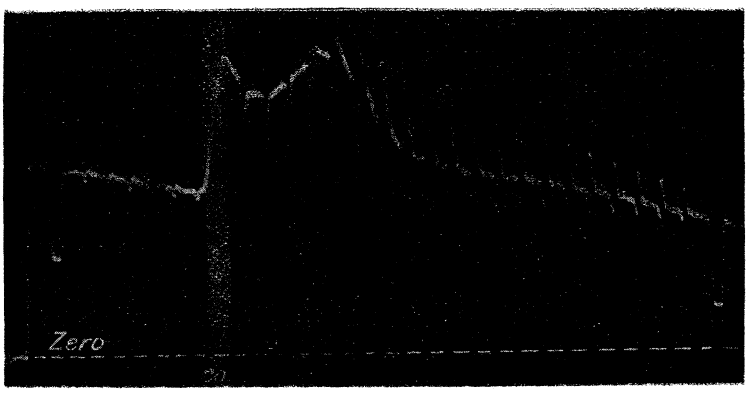
Fig. 66. (Obs. 2177.)

Fig. 65.—Record by the dead-beat galvanometer of an E.M.F. of 0·001 volt through a nerve, first in one, then in the opposite direction; time of closure = $\frac{1}{2}$ minute.

To show the falling time (= 10 to 12 seconds), and that the galvanometer is sufficiently dead-beat to permit us to take an excursion above the line, as in Plate 2177, to be significant of positive after-effect.

Fig. 66.—February 10, 1896.—Nerve in the late first or early second stage. To show the progressive diminution of the positive after-deflection.

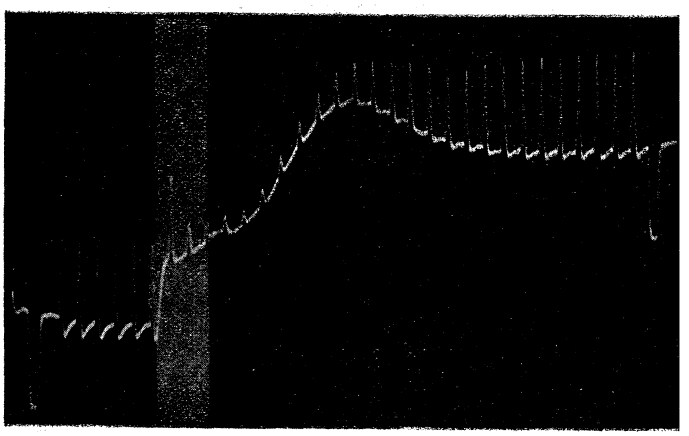
Fig. 67. (Obs. 2249.)



Record by dead-beat galvanometer of the effect of carbon dioxide on a very small negative variation (Connections as in fig. 3.)

This figure exhibits a point that has not been commented upon in the text, but is frequently witnessed in prolonged experiments, viz., a greatly augmented positive after-effect subsequent to the primary or secondary increase of the negative effect.

Fig. 68. (Obs. 2169.)



Record by dead-beat galvanometer of the effect of ether upon anelectrotonus. (Connections as in fig. 48, D.)

(NOTE.—Figs. 63–70 were added July 30, 1896.)

Fig. 69.

(3180, T.)

(3181, M.)

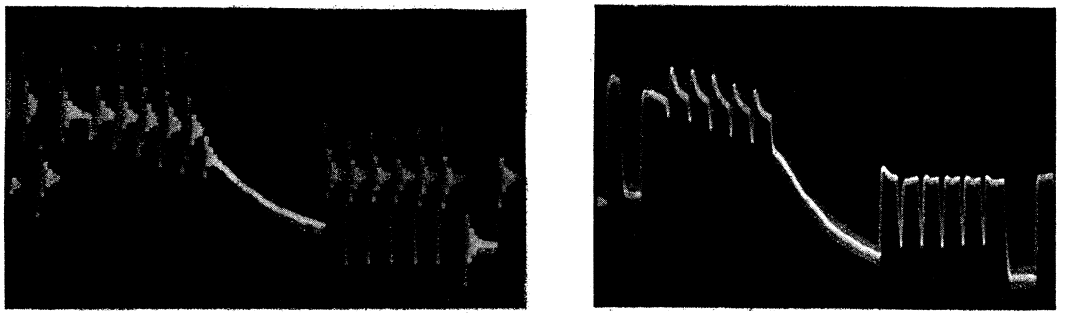
Simultaneous record of an experiment on the partially damped and fully damped galvanometers in circuit. The former, 3180, T., is shunted to $\frac{1}{4}$, oscillates 5 times per minute, and with the closure time of 7·5 seconds gives a swing-back = $\frac{2}{3}$rds the original swing. The latter, 3181, M., is not shunted, and has a falling-time of 12 seconds.

The two records, which exhibit the ordinary result of prolonged tetanisation, are given in explanation of the correction to be made for oscillation (3180, T.). In 3181, M., the closure-time being shorter than the falling-time, the negative variations do not reach their maximum. In 3180, T., the net after-effect is due to the state of nerve during the 2 to 3 seconds immediately after the $7\frac{1}{2}$ seconds excitation; in 3181, M., any positive after-effect would be due to the state of nerve during the last $\frac{2}{3}$rds of each minute. Such positive after-effect is better seen in plate 2177 (fig. 3B).

	Time.	I.	II.	(·6 I.)	II. net.	I./II.	0·001 volt.
	minutes.						
3180, T.	1	− 3·5	+5·7	(2·1)	+3·6	..	9·5
	2	− 5·3	+5·7	(3·2)	+2·5		
	3	− 6·0	+5·7	(3·6)	+2·1		
	5	− 6 0	+5·5	(3·6)	+1·9		
	12	− 6·5	+5·0	(3·9)	+1·1		
		Tetanisation, 10 units, 5 minutes $\alpha = -6{\cdot}5$ $\omega = +11{\cdot}5$					
	19	− 9·5	+5·5	(5·7)	−0·2		
	25	− 8·5	+5·0	(5·1)	−0·1		
	30	− 8·5	+5·0	(5·1)	−0·1		
	32	− 8·0	+5·0	(4·8)	−0·2	..	8·7
3181, M.	1	− 4	+4	..	..	..	13·5
	2	− 6	+2·5				
	3	− 6·5	+1·5				
	5	− 7	+1·2				
	12	− 7·5	+1				
		Tetanisation, 10 units, 5 minutes $\alpha = -10{\cdot}5$ $\omega = +17$					
	19	−11	0				
	25	−10	0				
	30	− 9·5	0				
	39	− 9	0	..	..	..	13·5

Fig. 70.

3188, T. 3189, M.



Simultaneous record of an experiment (influence of tetanisation on nerve in the second stage) on the partially damped and fully damped galvanometers in circuit.

	Time.	I.	II.	(·57 I.)	II. net.	I./II.	0·001 volt.
	minutes.						
3188, T.	0	− 3·2	+8·0	(1·8)	+6·2	..	15
	1	− 4·8	+9·4	(2·7)	+6·7		
	2	− 5·4	+9·6	(3·1)	+6·5		
	3	− 6·2	+9·5	(3·5)	+6·0		
	4	− 6·4	+9·4	(3·6)	+5·8		
		Tetanisation, 10 units, 5 minutes $\alpha = -6{\cdot}5$ $\omega = +13{\cdot}5$					
	11	−12	+4·0	(6·8)	−2·8		
	12	−12	+5·5	(6·8)	−1·3		
	13	−11·5	+5·3	(6·6)	−1·3		
	14	−11·5	+6·0	(6·6)	−0·6		
	15	−11·5	+6·5	(6·6)	−0·1	..	14·5
3189, M.	0	− 2	+4	..	..	..	14
	1	− 3·3	+3·5				
	2	− 3·8	+3·2				
	3	− 4·2	+2·8				
	4	− 4·5	+2·5				
		Tetanisation, 10 units, 5 minutes $\alpha = -4{\cdot}8$ $\omega = +11{\cdot}5$					
	11	− 9·3	0				
	12	− 9	0				
	13	− 9	0				
	14	− 8·8	0				
	15	− 8·5	0	..	..	..	13

TABLE D (continued).—Measurements of some of the Observations given in Tables B and C.

	Time.	I.	II.	($\frac{1}{3}$ I.)	II. net.	I./II.	0·001 volt.
	minutes.						
675 (fig. 29)	0	−13	+ 7·5	(4·3)	+ 3·2	4·1	26
		Tetanisation, 10 units, 5 minutes $\alpha = -13$ $\omega = +28$					
	6	−20	+12·5	(6·7)	+ 5·8	3·45	
	11	−16	+11	(5·3)	+ 5·7	2·8	
	16	−12	+ 7·5	(4)	+ 3·5	3·4	
	21	−10	+ 6·5	(3·3)	+ 3·2	3	24
676	0	−10	+10	(3·3)	+ 6·7	1·5	8
		Tetanisation, 10 units, 10 minutes $\alpha = -10$ $\omega = +19$					
	11	−14·5	+12·5	(4·8)	+ 9·7	1·5	
678	0	−10	+20	(3·3)	+16·7	0·60	18
		Tetanisation, 10 units, 10 minutes $\alpha = -10$ $\omega = +31·5$					
	11	−16	+21·5	(5·3)	+16·2	0·99	
	21	−13	+21·5	(4·3)	+17·2	0·76	
	31	−11	+16·5	(3·7)	+12·8	0·86	
	41	− 9	+14·5	(3)	+11·5	0·77	17
680	0	− 8·5	+18	(2·7)	+15·3	0·55	21
		Tetanisation, 10 units, 10 minutes $\alpha = -9$ $\omega = +29$					
	11	−16	+21	(5·3)	+15·7	1·02	
	21	−14	+20·5	(4·7)	+15·8	0·88	
	31	−10	+18	(3·3)	+14·7	0·68	
	40	− 9	+15·5	(3)	+12·5	0·72	18
689	0	−10	+14	(3·3)	+11·7	0·85	22
		Tetanisation, 10 units, 20 minutes $\alpha = -9$ $\omega = +27$					
	21	−18	+17·5	(6)	+11·5	1·57	
	26	−16	+14·5	(5·3)	+ 9·2	1·74	
	31	−15	+14	(5)	+ 9	1·67	
	36	−14	+14	(4·7)	+ 9·3	1·51	19

TABLE D (continued).—Measurements of some of the Observations given in Tables B and C.

	Time.	I.	II.	(⅓ I.)	II. net.	I./II.	0·001 volt.
	minutes.						
710 (fig. 35)	0	− 7	+19	(2·3)	+16·7	0·42	13
			CO_2 for 1 minute				
	2	−14	+ 6	(4·7)	+ 1·3	10·7	
	7	−12	+ 4	(4)	+ 0		
	12	−22	+28	(7·3)	+20·7	1·05	11·5
711	0	− 2·5	+18·5	(0·8)	+17·7	0·14	11
	4	− 3·5	+17·5	(1·2)	+16·3	0·21	
	5	−15	+ 7	(5)	+ 2	7·5	
	7	− 7·5	+ 2·5	(2·5)	+ 0		
CO_2	9	− 8	+ 2	(2·7)	− 0·7		
	11	− 8·5	+ 3	(2·8)	+ 0·2		
	14	− 9	+ 3	(3)	0		
	15	− 9·5	+ 4	(3·2)	+ 0·8	12	
	17	−16	+ 7·5	(5·3)	+ 2·2	7·3	
	20	−32	+17·5	(10·7)	+ 6·8	4·7	
	23	−27·5	+27·3	(9·2)	+18·1	1·3	10
737	0	− 2	+ 6	(0·7)	+ 5·3	0·38	
			Tetanisation, 10 units, 5 minutes				
	6	− 7	+ 9·5	(2·3)	+ 7·2	0·98	
	7	− 7	+12	(2·3)	+ 9·7	0·72	
	8	− 8	+13·5	(2·7)	+10·8	0·74	
741	0	−24	+17·5	(8)	+ 9·5	2·5	12
			Tetanisation, 10 units, 5 minutes $\alpha = -25$ $\omega = +39$				
	6	−29	+20·5	(9·7)	+10·8	3·7	
	11	−28	+20	(9·3)	+10·7	3·8	
	16	−27	+19	(9)	+10	2·7	
745	1	− 4	+ 5	(1·3)	+ 3·7	..	27
	4	− 4	+ 5	(1·3)	+ 3·7		
	5	−14	+ 2	(4·7)	− 2·7		
CO_2	6	− 3·5	0	(1·2)	− 1·2		
	7	− 2	0	(0·7)	− 0·7		
	8	− 3·5	0	(1·2)	− 1·2		
	9	− 7·5	0	(2·5)	− 2·5		
	10	−15	0	(5)	− 5		
	11	−22·5	+ 7	(7·5)	− 0·5		
	12	−27·5	+ 8	(9·2)	− 1·2		
	13	−28	+11	(9·3)	+ 1·7		
	14	−25·5	+15	(8·5)	+ 6·5		
	15	−23	+17	(7·7)	+ 9·3		
	16	−20	+19	(6·7)	+12·3		
	17	−19	+21	(6·3)	+14·7		

TABLE D (continued).—Measurements of some of the Observations given in Tables B and C.

	Time.	I.	II.	($\frac{1}{3}$ I.)	II. net.	I./II.	0·001 volt.
	minutes.						
747	0	− 4·5	+ 4·5	(1·5)	+ 3	..	15
	5	− 3·5	+ 4	(1·2)	+ 2·8		
	10	− 3	+ 3·5	(1)	+ 2·5		
CO_2	11	− 2	+ 2	(0·7)	+ 1·3		
	12	− 2	+ 2	(0·7)	+ 1·3		
	13	− 5	+ 7	(1·7)	+ 5·3		
	14	−12	+ 7	(4)	+ 3		
	19	−17·5	+11	(5·8)	+ 5·2		
	20	−17·5	+10	(5·8)	+ 4·2		
CO_2	21	−12	0	(4)	− 4		
	22	− 9	0	(3)	− 3		
	23	− 7·5	0	(2·5)	− 2·5		
	24	− 8	0	(2·7)	− 2·7		
	25	−10	0	(3·3)	− 3·3		
	30	−24	+ 9	(8)	+ 1		
	35	−14	+ 9	(4·7)	+ 4·3		
	40	− 7	+ 6	(2·3)	+ 3·7	..	14
751	0	+ 2	+ 9	(0·67)	+ 9·67		
		Tetanisation, 10 units, 5 minutes $\alpha = +2$ $\omega = +9$					
	6	− 5	+ 9·5	(1·67)	+ 7·83		
	11	− 4·5	+ 9	(1·5)	+ 7·5		
	16	− 4	+ 8	(1·33)	+ 6·7		
	21	− 3·5	+ 8	(1·2)	+ 6·8		
752	0	− 3	+ 8	(1)	+ 7		
	7	− 3	+ 8	(1)	+ 7		
		CO_2 for 1 minute					
	10	− 0·5	0	(0·2)	− 0·2		
	11	− 1	0	(0·3)	− 0·3		
	12	− 3·5	0	(1·2)	− 1·2		
	13	− 8·5	+ 3·5	(2·8)	+ 0·7		
	14	−15	+ 7·5	(5)	+ 2·5		
	15	−15	+ 8·5	(5)	+ 3·5		
	20	− 7·5	+12	(2·5)	+ 9·5		
	25	− 6·5	+ 8	(2·2)	+ 5·8		
	30	− 5	+ 7·5	(1·8)	+ 5·7		
761	0	− 7	+ 4	(2·3)	+ 1·7	4·1	26
		Tetanisation, 10 units, 5 minutes $\alpha = -7$ $\omega = +14$					
	6	−11	+ 6	(3·7)	+ 2·3	4·8	
	11	−10	+ 5	(3·3)	+ 1·7	5·9	
	16	− 9	+ 4	(3)	+ 1	9·0	26

TABLE D (continued).—Measurements of some of the Observations given in Tables B and C.

	Time.	I.	II.	($\frac{1}{3}$ I.)	II. net.	I./II.	0·001 volt.
762	minutes. 0	− 4·5	+11·5	(1·5)	+10	0·45	22
		Tetanisation, 10 units, 5 minutes $\alpha = -4{\cdot}5$ $\omega = +24$					
	6	−12	+13·5	(4)	+ 9·5	1·26	
	11	−10	+12	(3·3)	+ 8·7	1·13	
	16	− 9	+11·5	(3)	+ 8·5	1·06	
782	0	+11	− 1·5	(3·7)	+ 1·2		25
	4	+11	− 1·5	(3·7)	+ 1·2		
		Tetanisation, 10 units, 5 minutes $\alpha = +11$ $\omega = -11$					
	10	+11	− 3·5	(3·7)	+ 0·2		
	15	+11·5	− 1·0	(3·8)	+ 2·8		
	20	+12·5	− 1·0	(4·2)	+ 3·2		23

	Time.	I.	II.	($\frac{2}{3}$ I.)	II. net.	I./II.	0·001 volt.
857	minutes. 0	− 2	+ 8	(1·3)	+ 6·7	3·3	15·5
		Tetanisation, 5 minutes, 10 units $\alpha - 2$ $\omega + 21{\cdot}5$					
	6	−12·5	+10	(8·5)	+ 1·5	8·3	
	11	− 8	+ 9·5	(5·4)	+ 2·6	3·1	
	16	− 5·5	+ 8·5	(3·7)	+ 1·8	3	
858 (fig. 30)	0	+ 7	+ 1	(4·7)	+ 5·7	..	13·5
		Tetanisation, 5 minutes, 10 units $\alpha + 5$ $\omega + 17$					
	6	− 9	+ 8	(6)	+ 2	4·7	
	11	− 4·5	+ 7	(3)	+ 4	1·1	
	16	− 1·5	+ 5	(1)	+ 4	·37	
859 (fig. 31)	0	+ 8	− 1	(5·4)	+ 4·4		
		CO_2, 3 minutes					
	10	− 9	+10	(6)	+ 4	2·3	
	15	− 2·5	+ 8·5	(1·7)	+ 6·8	0·3	

TABLE D (continued).—Measurements of some of the Observations given in Tables B and C.

	Time.	I.	II.	($\frac{2}{3}$ I.)	II. net.	I./II.	0·001 volt.
	minutes.						
869	0	− 7	+ 4·5	(4·7)	−0·2		
(fig. 16)	5	− 7	+ 4·5	(4·7)	−0·2		
CO_2	6	− 7	(?)				
	7	−13	+ 8·5	(8·7)	−0·2		
	8	−14·5	+ 6·5	(9·7)	−3·2		
	9	−14·5	+ 7·0	(9·7)	−2·7		
	10	−15·5	+ 8·0	(10·3)	−2·3		
	11	−16	+ 9·0	(10·7)	−1·7		
	12	−17	+10·0	(11·3)	−1·3		
	13	−17·5	+10·5	(11·7)	−1·2		
	14	−18	+12·0	(12)	0		
	15	−17·5	+12·0	11·7	+0·3		
	20	−15	+11·5	10	+1·5		
	25	−12	+ 9·5	8	+1·5		
874	0	+15	−11	(10)	−1	..	11
	5	+15	−10	(10)	0		
		Tetanisation, 10 units, 5 minutes $\alpha = +15$ $\omega = -7$					
	11	+12	− 5	(8)	+3		
	16	+11	− 5	(7·3)	+2·3		
	21	+11·5	− 6	(7·7)	+1·7		
	26	+12	− 6·5	(8)	+1·5		
905	0	+12	− 7	(8)	+1	..	16
(fig. 36)	8	+11	− 7	(7·3)	+0·3		
		Tetanisation, 10 units, 10 minutes $\alpha = +11·5$ $\omega = -15·5$					
	20	+14	− 9·5	(9·3)	−0·2		
	30	+13	− 8	(8·7)	−0·7		
	40	+13	− 8	(8·7)	−0·7		
	50	+12	− 7·5	(8)	+0·5	..	14
912	0	− 7	+13	(4·7)	+8·3	..	14
(fig. 37)	11	− 7·5	+13	(5)	+8		
		Tetanisation, 10 units, 10 minutes $\alpha = -9$ $\omega = +11$					
	22	− 5	0	(3·3)	+3·3		
	30	− 8	+ 6·5	(5·3)	+1·2		
	40	−10·5	+ 9·5	(7)	+2·5		
	50	−10	+ 9·5	6·7	+2·8	..	12·5

TABLE D (continued).—Measurements of some of the Observations given in Tables B and C.

	Time.	I.	II.	(⅔ I.)	II. net.	I./II.	0·001 volt.
	minutes.						
928	0	− 8·5	+ 9·5	(5·65)	+3·85	2·2	19·5
		Tetanisation, 5 minutes, 10 units α − 8 ω + 43					
	6	−31	+26	(20·7)	+5·3	5·85	
	11	−21	+19	(14)	+5	4·2	
	16	−14·5	+14·5	(9·35)	+5·15	2·8	
	21	−10	+10	(6·7)	+3·3	3	
929	0	− 1	+ 3·5	(·67)	+2·83	1·23	18
		Tetanisation, 5 minutes, 10 units α − 1 ω + 26·5					
	6	−18·5	+15	(12·3)	+2·7	6·9	
	11	− 9	+ 8·5	(6)	+2·5	3·6	
	16	− 4	+ 5·5	(2·7)	+2·8	1·4	
	21	− 1·5	+ 1·5	(1)	+ ·5	3	
933 (fig. 40)	0	+ 7	−10	(4·7)	−5·3	..	17·5
		Tetanisation, 5 minutes, 10 units α + 6·5 ω + 30					
	6	−15·5	+16	(10·3)	+5·7		
	11	− 5·5	+ 5	(3·7)	+1·3		
	16	+ 1·5	− 7	(1)	−6		
	21	+ 4·5	− 9	(3)	−6		
935	0	− 7	+ 8	4·7	+3·3	..	5·5
	5	− 6·5	+ 7·5	4·3	+3·2		
CO_2	6	− 5·5	+ 8	3·7	+4·3		
	7	− 8·5	+ 7·5	5	+2·5		
	8	−11·5	+ 4·5	7·7	−3·2		
	9	−10·5	+ 2·5	7	−4·5		
	10	− 8·5	+ 1·5	5·7	−4·5		
	11	− 6·5	+ 1	4·3	−3·3		
	12	− 7	+ 1·5	4·7	−3·2		
	13	− 9	+ 3·5	6	−2·5		
	14	−13·5	+ 6·5	9	−2·5		
	15	−17	+ 9·5	11·3	−1·8		
	20	−11	+11	7·3	+3·7		
	25	− 8·5	+10·5	5·7	+4·8		
937	0	− 6	+ 6	(4)	+ 2	3	10·5
		Tetanisation, 10 units, 5 minutes α = − 7 ω = + 21					
	6	−11	+ 8	(7·3)	+0·7	15·7	
	11	− 8·5	+ 7	(5·7)	+1·3	6·5	
	16	− 8	+ 6·5	(5·3)	+1·2	5·4	
	21	− 7	+ 6·5	(4·7)	+1·8	3·6	

TABLE D (continued).—Measurements of some of the Observations given in Tables B and C.

	Time.	I.	II.	($\frac{2}{3}$ I.)	II. net.	I./II.	0·001 volt.
942	minutes. 0	+11	− 8	(7·3)	− 0·7	11·4	35
		Tetanisation, 10 units, 5 minutes $\alpha = +11$ $\omega = -23·5$					
	6	+13·5	−16	(9)	− 7	1·74	
	11	+11·5	−10·5	(7·7)	− 2·8	4·1	
	16	+11·5	− 9·5	(7·7)	− 1·8	6	
958	0	− 3	+13·5	(2)	+11·5	0·26	23·5
	1	− 6·5	+15	(4·3)	+10·7	0·61	
	2	− 8·5	+15	(5·7)	+ 9·3	0·91	
	3	− 9·5	+15	(6·3)	+ 8·7	1·09	
	4	−10	+15	(6·7)	+ 8·3	1·20	
	5	−11	+15	(7·3)	+ 7·7	1·41	
	6	−11·5	+15	(7·7)	+ 7·3	1·58	
	7	−11·5	+15	(7·7)	+ 7·3	1·58	
	8	−11·5	+15	(7·7)	+ 7·3	1·58	
		Tetanisation, 10 units, 5 minutes $\alpha = -12$ $\omega = +36$					
	14	−26	+16·5	(17)	− 0·5		
	19	−22	+16	(15)	+ 1·0	22	
		Pause of 5 minutes					
	25	−13	+14	(8·6)	+ 5·4	2·4	
	30	−15	+15	(10)	+ 5	3	
960 (fig. 42)	1	+ 5	+15	(3·3)	+18·3	..	38
	5	0	+14	0			
		Tetanisation, 10 units, 5 minutes $\alpha = 0$ $\omega = +45·5$					
	12	−34	+19·5	22·7	− 3·2		
	15	−30	+17·5	20	− 2·5		
	20	−25	+17	16·7	+ 0·3		
	25	−21·5	+17	14·3	+ 2·7	..	38
982 (fig. 26)	0	+14	− 8·5	(9·3)	+ 0·8	..	19·5
	5	+11·5	− 8	(7·7)	− 0·3		
		CO_2 for 1 minute					
	6	+12	− 7	(8)	+ 1·0		
	7	+ 8	− 4	(5·3)	+ 1·3		
	8	+ 7	− 2·5	(4·7)	+ 2·2		
	10	+ 5	− 1·5	(3·3)	+ 1·8		
	14	+ 7	− 2	(4·7)	+ 2·7		
	20	+ 9	− 3·5	(6·0)	+ 2·5		
	25	+ 9·5	− 4·5	(6·3)	+ 1·8		
	29	+10	− 5·5	(6·7)	+ 1·2		

TABLE D (continued).—Measurements of some of the Observations given in Tables B and C.

	Time.	I.	II.	(⅔ I.)	II. net.	I./II.	0·001 volt.
	minutes.						
983	0	+11·5	− 7	(7·7)	+ 0·7	..	19·5
	5	+11·5	− 7	(7·7)	+ 0·7		
	6	0	0				
CO_2	7	0	0				
	8	− 2	0	(1·3)	+ 1·3		
	9	− 2	0	(1·3)	+ 1·3		
	12	+ 3·5	− 5	(2·3)	− 2·7		
	13	− 3	?				
	14	− 4	+ 3	(2·7)	+ 0·3		
	15	− 5	+ 4	(3·3)	+ 0·7		
	16	− 5·5	+ 5·5	(3·7)	+ 1·8		
	17	− 7	+ 7	(4·7)	+ 2·3		
	18	− 5·5	+ 7·5	(3·7)	+ 3·8		
	19	− 3·5	+ 7	(2·3)	+ 4·7		
	20	0	+ 6	(0)	+ 6·0		
	21	+ 2	+ 5	(1·3)	+ 6·3		
	22	+ 3·5	+ 4·5	(2·3)	+ 6·8		
	23	+ 5·5	+ 4·5	(3·7)	+ 8·2		
	24	+ 6·5	+ 4·5	(4·3)	+ 8·8		
	25	+ 7·5	+ 5	(5)	+10		
984 (fig. 32)	0	+14·5	− 6·5	(9·7)	+ 3·2	..	19
	5	+13·5	− 7	(9)	+ 2		
		Tetanisation, 10 units, 5 minutes $\alpha = +13·5$ $\omega = -2·5$					
	11	+ 5·5	− 2·5	(3·7)	+ 1·2		
	16	+ 9·5	− 4·5	(6·3)	+ 1·8		
	21	+10·5	− 6·5	(7)	+ 0·5		
	25	+12	− 7·5	(8)	+ 0·5		
985	0	+14·5	− 8·5	(9·7)	+ 1·2	..	19
	5	+14·5	− 8·5	(9·7)	+ 1·2		
		Tetanisation, 10 units, 5 minutes $\alpha = +14·5$ $\omega = -6·5$					
	11	+ 8·5	− 6	(5·7)	− 0·3		
	16	+10·5	− 7	(7)	0		
	21	+12	− 8	(8)	0		
990	0	+21	−13	(14)	+ 1	..	54
		CO_2 for 1 minute					
	1	+20	−10	(13·3)	+ 3·3		
	5	+ 1·5	+ 5	(1)	+ 4		
	10	+ 5·5	+ 5·5	(3·7)	+ 1·8		
	15	+12	− 4	(8)	+ 4		
	20	+14	− 6	(9·3)	+ 3·3		

TABLE D (continued).—Measurements of some of the Observations given in Tables B and C.

	Time.	I.	II.	(⅔ I.)	II. net.	I./II.	0·001 volt.
	minutes.						
991	0	− 4·5	+ 9	(3)	+ 6	..	23
		Tetanisation, 10 units, 5 minutes $\alpha = -4{\cdot}5$ $\omega = +12$					
	6	− 5·5	+ 9·5	(3·7)	+ 5·8		
	11	− 5	+10	(3·3)	+ 6·7		
	16	− 4·5	+10	(3)	+ 7		
	21	− 3·5	+ 9·5	(2·3)	+ 7·2		
993	0	+ 9·5	− 7	(6·3)	− 0·7	..	19
	5	+ 7·5	− 7	(5)	− 2		
		CO_2 for 1 minute					
	6	−15	+ 9·5	(10)	− 0·5		
	7	− 8	+ 3	(5·3)	− 2·3		
	8	−11	+ 5	(7·3)	− 2·3		
	9	−16·5	+10	(11)	− 1		
	10	−21	+14	(14)	0		
	15	−25	+26·5	(16·7)	+ 9·8		
	20	−15	+21	(10)	+11		
	25	− 7·5	+15·5	(5)	+10·5		
994	0	0	+ 9	(0)	+ 9	..	17·5
	3	0	+ 9	(0)	+ 9		
		Tetanisation, 10 units, 5 minutes $\alpha = 0$ $\omega = +14$					
	9	− 3·5	+ 8	(2·3)	+ 5·7		
	13	− 3	+ 8	(2)	+ 6		
	18	− 2·5	+ 7·5	(1·7)	+ 5·8		
	23	− 2·5	+ 7	(1·7)	+ 5·3		
2003	0	− 7·5	+ 3	(5)	− 2	..	37
		Tetanisation, 10 units, 5 minutes $\alpha = -8{\cdot}5$ $\omega = +5$					
	6	− 8·5	+ 3·5	(5·7)	− 2·2		
	20	− 9·5	+ 3	(6·3)	− 3·3		
2004	0	− 2·5	+10·5	(1·7)	+ 8·8		
	5	− 4	+11	(2·7)	+ 8·3		
		Tetanisation, 10 units, 5 minutes $\alpha = -4{\cdot}5$ $\omega = +6{\cdot}5$					
	11	− 2·5	+ 2·5	(1·7)	+ 0·8		
	16	− 5	+12	(3·3)	+ 8·7		
	21	− 7	+11·5	(4·7)	+ 6·8		
	26	− 6·5	+11	(4·3)	+ 6·7		

TABLE D (continued).—Measurements of some of the Observations given in Tables B and C.

	Time.	I.	II.	($\frac{2}{3}$ I.)	II. net.	I./II.	0·001 volt.
2005	minutes. 0	− 8	+15	(5·3)	+10·7		
	4	− 7	+17	(4·7)	+12·3		
		Tetanisation, 10 units, 5 minutes $\alpha = -7$ $\omega = +9·5$					
	5	− 3·5	+ 4	(2·3)	+ 1·7		
	10	− 7·5	+15·5	(5)	+10·5		
	15	−10	+15	(6·7)	+ 8·3		
2006	0	− 4·5	+ 8	(3)	+ 5		
	5	− 4·5	+ 8	(3)	+ 5		
	10	− 4·5	+ 8	(3)	+ 5		
		CO_2 for 1 minute					
	11	−10	+ 6	(6·7)	− 0·7		
	12	− 4·5	+ 1	(3)	− 2		
	13	− 2·5	0	(1·7)	− 1·7		
	14	− 4·5	+ 1	(3)	− 2		
	15	− 6·5	+ 3·5	(4·3)	− 0·8		
	20	−10	+ 9	(6·7)	+ 2·3		
	28	− 7·5	+ 7	(5)	+ 2		
2054	0	− 8	+ 5	(5·3)	− 0·7	..	13
	5	−10·5	+ 5	(7)	− 2·0		
		Tetanisation, 10 units, 5 minutes $\alpha = -10·5$ $\omega = +17$					
	11	−14·5	+ 5	(9·7)	− 4·7		
	20	−14	+ 4	(9·3)	− 5·3		
	28	−13·5	+ 4	(9)	− 5		
2055	0	−12	+ 4	(8)	− 4	..	15
	5	−12·5	+ 4	(8·3)	− 4·3		
		Tetanisation, 10 units, 5 minutes $\alpha = -12$ $\omega = +17$					
	11	−16·5	+ 4	(11)	− 7		
	20	−14·5	+ 3·5	(9·7)	− 6·2		
	27	−14	+ 2·5	(9·3)	− 6·8		
2057	0	− 5	+ 7	(3·3)	+ 3·7	..	16
	10	−12	+ 8	(8)	0		
		Tetanisation, 10 units, 5 minutes $\alpha = -11$ $\omega = +45$?					
	27	−23	+19	(15·3)	+ 3·7		
	30	−22	+17	(14·7)	+ 2·3		
	40	−18	+14	(12)	+ 2		
	50	−17	+13	(11·3)	+ 1·7		

TABLE D (continued).—Measurements of some of the Observations given in Tables B and C.

	Time.	I.	II.	(⅔ I.)	II. net.	I./II.	0·001 volt.
2068	minutes. 1	− 6	+14	(4)	+14	..	11
	10	− 8·5	+16	(5·7)	+11·3		
		Tetanisation, 10 units, 5 minutes					
	15	−16	+15	(10·7)	+ 4·3		
	25	−16	+14	(10·7)	+ 3·3		
		Tetanisation, 100 units, 5 minutes					
	30	− 3	+ 3·5	(2)	+ 1·5		
	40	− 7·5	+10	(5)	+ 5		
		Tetanisation, 10 units, 5 minutes					
	45	−19	+19	(12·7)	+ 6·3		
	55	−18	+18	(12)	+ 6		
		Tetanisation, 100 units, 5 minutes					
	60	− 1	+ 1·5	(0·7)	+ 0·8		
	70	− 2·5	+ 3·5	(1·7)	+ 1·8	..	8·5
2069	1	−10	+ 9	(6·7)	+ 2·3	..	21
	10	−12	+11	(8)	+ 3		
		Tetanisation, 10 units, 5 minutes					
	15	−20	+18	(13·3)	+ 4·7		
	25	−16·5	+14·5	(11)	+ 3·5		
		Tetanisation, 100 units, 5 minutes					
	30	− 3	+ 2·5	(2)	+ 0·5		
	40	− 6·5	+ 5·5	(4·3)	+ 1·2		
		Tetanisation, 10 units, 5 minutes					
	45	−14	+11·5	(9·3)	+ 2·2		
	55	−12	+10·5	(8)	+ 2·5		
		Tetanisation, 100 units, 5 minutes					
	60	0	0				
	70	0	0	..	..	..	19·5
2072	0	+ 1·5	+ 4	(1)	+ 5	..	15
		Tetanisation, 10 units, 5 minutes $\alpha = +1·5$ $\omega = +14$					
	6	− 3	+ 7	(2)	+ 5		
	10	0	+ 5·5	(0)	+ 5·5		
	15	0	+ 5	(0)	+ 5		
	20	+ 1	+ 4	(0·7)	+ 4·7		

TABLE D (continued).—Measurements of some of the Observations given in Tables B and C.

	Time.	I.	II.	(⅔ I.).	II. net.	I./II.	0·001 volt.
	minutes.						
2073	0	+ 7·5	+ 8·5	(5)	+13·5	..	30
	5	+ 7·5	+10·5	(5)	+13·5		
			CO_2 for 2 minutes				
	9	−16	0	(10·7)	−10·7		
	10	−17	?	(11·3)	?		
	20	−44	+44	(29·3)	+14·7	..	30
	27	−16	+26	(10·7)	+15·3		
2074	1	+ 3	+ 8·5	(2)	+10·5	..	19
	5	+ 1·5	+ 8·5	(1)	+ 9·5		
			Tetanisation, 10 units, 5 minutes $\alpha = +1·5$ $\omega = +18$				
	11	− 9·5	+11·5	(6·3)	+ 5·2		
	15	− 4·5	+ 8·5	(3)	+ 5·5		
	20	0	+ 7	(0)	+ 7		
2075	0	+ 5·5	+ 4	(3·7)	+ 7·7	..	19
	4	+ 6	+ 4	(4)	+ 8		
			CO_2 for 2 minutes				
	6	− 7	0	(4·7)	+ 4·7		
	10	−11	+ 4	(7·3)	+ 3·3		
	12	−23	+15·5	(15·3)	+ 0·2		
	15	−19·5	+17	(13)	+ 4·0		
	20	− 9	+11	(6)	+ 5		
	25	0	+ 5·5	(0)	+ 5·5	..	19
2080	0	− 3	+ 6	(2)	+ 4	..	12·5
	4	− 3·5	+ 5·5	(2·3)	+ 3·2		
			CO_2 for 2 minutes				
	7	−16·5	+ 9	(11)	− 2		
	10	−16·5	+ 9	(11)	− 2		
	12	−17·5	+12·5	(11·7)	+ 0·8		
	15	−12	+14	(8)	+ 6		
	20	− 8	+14	(5·3)	+ 8·7		
	25	− 6·5	+12	(4·3)	+ 7·7	..	12
2081	0	+ 5·5	− 4	(3·7)	− 0·3		
			Tetanisation, 10 units, 5 minutes				
	7	+10·5	− 3·5	(7)	+ 3·5		
	12	+ 8·5	− 4·5	(5·7)	+ 1·2		
	17	+ 8	− 5	(5·3)	+ 0·3		
	22	+ 7·5	− 5	(5)	0		

TABLE D (continued).—Measurements of some of the Observations given in Tables B and C.

	Time.	I.	II.	(⅔ I.).	II. net.	I./II.	0·001 volt.
2082	minutes. 0	+ 7·5	− 4·5	(5)	+ 0·5	..	18
	5	+ 6	− 5	(4)	− 1·0		
CO_2	6	− 8·5	+ 3·5	(5·7)	− 2·2		
	7	− 7·5	+ 2	(5)	− 3·0		
	8	− 5	+ 1·5	(3·3)	− 1·8		
	9	− 3	+ 1	(2)	− 1·0		
	10	− 3	+ 1	(2)	− 1·0		
	12	− 5	+ 3·5	(3·3)	+ 0·2		
	15	− 6·5	+10	(4·3)	+ 5·7		
	20	− 4	+ 5	(2·7)	+ 2·3		
2084	0	− 8·5	+ 6·5	5·7	+ 0·8		
	3	−10·5	+ 6	7	− 1		
		Tetanisation, 100 units, 5 minutes $\alpha = -20{\cdot}5$ $\omega = +15$					
	10	?	?				
	15	− 2	+ 2	1·3	+ 0·7		
	20	− 4	+ 2·5	2·7	− 0·2		
	25	− 5·5	+ 3	3·7	− 0·7	..	13
2085	1	− 7·5	+10	(5·0)	+ 5	..	27
	4	− 8·0	+ 8·5	(5·3)	+ 3·2		
		Tetanisation, 10 units, 5 minutes $\alpha = ?$ $\omega = +4{\cdot}5$					
	9	− 6·5	+ 3	(4·3)	− 1·3		
	15	− 9·0	+ 4·5	(6·0)	− 1·5		
	20	− 8·5	+ 5·5	(5·7)	− 0·2		
	25	− 7·5	+ 5·5	(5·0)	+ 0·5		
2241	1	− 2·5	0				
	2	− 2·5	0				
	3	− 2·5	0				
CO_2	4	− 5	0				
	5	−17	0				
	6	−22	+ 1				
	7	−25	+ 2				
	8	−26	+ 2·5				
	9	−24	+ 3·5				
	10	−20	+ 6				

TABLE D (continued).—Measurements of some of the Observations given in Tables B and C.

	Time.	I.	II.	(⅔ I.)	II. net.	I./II.	0·001 volt.
	minutes.						
2242	1	− 6·5	+16			..	12
	2	− 5	+15				
	3	− 4·5	+14				
	4	−22	0				
	5	−19	0				
CO_2	6	−15	0				
	7	−12·5	0				
	8	−11	0				
	9	−11	0				
	10	−14	0				
	11	−18·5	0				
	12	−20	0				
	13	−21	+ 1·5				
	14	−18	+ 3				
	15	−14	+ 6·5				
	16	−10·5	+ 8·5				
	17	− 6	+11				
	18	− 4	+11				
	19	− 2·5	+10·5				
	20	− 1	+10				
	21	0	+ 9				

	Time.	I.	II.	(·6 I.)	II. net.	I./II.	0·001 volt.
	minutes.						
200	0	−10	+ 7	(6·0)	+ 1·0		
(fig. 14)	5	− 9·5	+ 7	(5·7)	+ 1·3		
	6	− 9·5	+ 6	(5·7)	+ 0·3		
	7	− 9	+ 4·5	(5·4)	− 0·9		
	8	0	0	(0)	0		
	9	0	0	(0)	0		
	10	− 6	+ 3	(3·6)	− 0·6		
	11	−14	+ 9	(8·4)	+ 0·6		
	12	−18	+13	(10·8)	+ 2·2		
	13	−19	+14·5	(11·4)	+ 3·1		
	14	−18	+14	(10·8)	+ 3·2		
	15	−16	+12·5	(9·6)	+ 2·9		
	20	−12·5	+ 9	(7·5)	+ 1·5		
	25	−11	+7·5	(6·6)	+ 0·9		

	Time.	I.	II.	(⅔ I.)	II. net.	I./II.	0·001 volt.
	minutes.						
309	0	−11·5	+ 9·5	7·7	+ 1·8	..	10
(fig. 4)	5	−11	+ 9·3	7·3	+ 2		
	10	−10·5	+ 8·7	7	+ 1·7		
	15	10	8·3	6·7	+ 1·6		
	20	9·5	7·7	6·3	+ 1·4		
	25	9	7·5	6	+ 1·5		
	30	3·5	7·2	5·7	+ 1·5		
	35	8·3	6·8	5·5	+ 1·3		
	40	8	6·8	5·3	+ 1·5		

TABLE D (continued).—Measurements of some of the Observations given in Tables B and C.

	Time.	I.	II.	($\frac{2}{3}$ I.)	II. net.	I./II.	0·001 volt.
	minutes.						
334	0	11·5	8·5	7·7	+ 0·8	..	16·5
(fig. 1)	5	11	8	7·3	+ 0·7		
	10	10·5	7·5	7	+ 0·5		
	15	10·5	7·5	7	+ 0·5		
	20	10	6·5	6·7	− 0·2		
	25	10	6	6·7	− 0·7	..	14·5
	Time.	I.	II.	(·6 I.)	II. net.	I./II.	0·001 volt.
	minutes.						
537	1	−14·5	+13·5	8·7			
(fig. 15)	2	−14·5	+13·5	8·7			
	3	−14·5	+13	8·7			
	4	−21	+23	12·6			
	5	−27·5	+14·5	16·8			
	6	−22	+10	13·2			
	7	−18·5	+ 9	11·0			
	8	−15·5	+ 8	9·6			
	9	−20	+11	12			
	10	−27	+16·5	16·2			
	11	−36	+23	21·6			
	17	−37	+33	22·3			
	21	−27	+27·5	16·2			
708	0	−17·5	+20	(10·5)	+ 9·5	..	20·5
(fig. 6)	10	−17·5	+21·5	(10·5)	+11·0		
	20	−17	+21·5	(10·2)	+11·3		
	30	−17	+21·5	(10·2)	+11·3		
	40	−17	+21·5	(10·2)	+11·3	..	17·5
2248	1	− 4	+ 1·5	..	..	..	11
	2	− 4	+ 1·5				
	3	−10	0				
	4	−13	0	CO_2			
	5	−12	0				
	6	−12	0				
	7	−13	0				
	8	−18	0				
	9	−23					
	10	−27					
	11	−27					
	12	26	+ 2				
	13	24	+ 2·5				
	14	21	+ 3				
	15	18	+ 3				
	16	16	+ 3				
	17	14	+ 3				
	18	12	+ 3				
	19	10	+ 3				
	20	9	+ 3				

TABLE D (continued).—Measurements of some of the Observations given in Tables B and C.

	Time.	I.	II.	($\frac{2}{3}$ I.)	II. net.	I./II.	0·001 volt.
	minutes.						
2249	1	− 1	+ 0·5	..	..	..	11·5
	2	− 1	+ 0·5				
	3	− 1	+ 0·5				
	4	− 1	+ 0·5				
	5	− 1	+ 0·5				
	6	− 1	+ 0·5				
	7	− 6	..	} CO_2			
	8	− 7·5					
	9	− 8					
	10	− 9·5					
	11	−12					
	12	−14	+ 2·5				
	13	−13	+ 4				
	14	− 9·5	+ 7·5				
	15	− 7	+ 9				
	16	− 5·5	+10·5				
	17	− 5	+10·5				
	18	− 4·5	+ 9·5				
	19	− 4	+ 8·5				
	20	− 3·5	+ 7·5				
	25	− 2	+ 4	..	..	..	11·5
2251	0	− 8	+ 5	..	..	..	10·5
	5	− 8	+ 5				
			CO_2 for 1 minute				
	7	−20	0				
	8	−19	0				
	9	−22	0				
	10	−28	+ 2				
	11	−34	+ 6				
	12	−34	+ 6				
	13	−32	+ 7				
	14	−29	+ 7·5				
	15	−27	+ 8·5				
	20	−18	+8	.	..	..	10
	24	−13·5	+7				

TABLE D (continued).—Measurements of some of the Observations given in Tables B and C.

	Time.	I.	II.	($\frac{2}{3}$ I.)	II. net.	I./II.	0·001 volt.
	minutes.						
2256	0	− 6	+ 1	..	..	..	13·5
	5	− 6	+ 1				
	6	− 6	0				
	7	− 8	0	CO_2			
	8	− 5	0				
	9	− 5	0				
	10	− 8	0				
	11	−12	+ 1				
	12	−16	+ 1·5				
	13	−17	+ 2				
	14	−16	+ 2				
	15	− 5	+ 2				
	16	− 3·5	0	CO_2			
	17	− 2·5	0				
	18	− 4·5	0				
	19	− 8	0				
	20	−13	+ 1				
	21	−17	+ 1·5				
	22	−18	+ 2·5				
	23	−16·5	+ 3				
	24	−15	+ 3				
	25	−13·5	+ 3				
	26	−12	+ 2·5				
	27	−10·5	+ 2·5				
	28	− 9·5	+ 2				
	29	− 8·5	+ 1·5				
	30	− 8	+ 1	..	..	..	13
2250	0	− 8·5	+ 3·5	..	..	..	11
	5	− 8·5	+ 2				
		Tetanisation, 10 units, 5 minutes $\alpha = -8{\cdot}5$ $\omega = +12$					
	11	−10	+ 1				
	15	− 9	+ 1				
	20	− 8	+ 1				
	25	− 7	+ 1				
2258	0	− 3·5	+ 2·5	..	..	..	12·5
	5	− 3·5	+ 2·5				
		Tetanisation, 5 minutes $\alpha = -3{\cdot}5$ $\omega = +8$					
	11	− 6	+ 1·5				
	15	− 5	+ 1·5				
	20	− 5	+ 1·5	..	..	..	11·5

TABLE D (continued).—Measurements of some of the Observations given in Tables B and C.

	Time.	I.	II.	($\frac{1}{3}$ I.)	II. net.	I./II.	0·001 volt.
	minutes.						
2262	0	−15	+ 5				
	5	−15	+ 5				
			Tetanisation, 5 minutes $\alpha = -15$ $\omega = +26$				
	11	−22	+ 3·5				
	15	−20	+ 3·5				
	20	−19	+ 3·5				
2266	0	− 6	+ 4	..	..	..	7·5
	5	− 6	+ 3				
			Tetanisation, 5 minutes $\alpha = -6$ $\omega = +10$				
	11	− 9	+ 1·5				
	15	− 7	+ 2				
	20	− 6	+ 2	..	..	..	5·5
2268	0	− 2·5	+ 1·5	..	..	..	4·5
	4	− 2·5	+ 1·5				
			CO_2 for 2 minutes.				
	10	−11	+ 2				
	15	− 8	+ 3·5				
	20	− 5	+ 2·5	..	..	..	5
2271	0	−13	+ 3·5	..	..	..	14
	6	−15	+ 3·5				
			Tetanisation, 5 minutes $\alpha = -15$ $\omega = +29$				
	12	−23·5	+ 3				
	17	−20	+ 3·5				
	22	−18·5	+ 3·5	..	..	..	11
2274	0	− 6	+ 1·5	..	..	..	10
	5	− 6·5	+ 1·5				
			CO_2 for 1 minute				
	6	− 8	0				
	7	−14	+ 2				
	8	−17	+ 3				
	9	−19	+ 4				
	10	−20	+ 4·5				
	15	−17	+ 4				
	20	−12·5	+ 3				
	24	−11	+ 2	..	..	..	12

TABLE E.—Data relating to Chapter IV., § 4. Action of CO_2 on Polarising and Electrotonic Currents, &c.

Effect of Et_2O upon neg. var., upon anelectrotonus, and upon katelectrotonus.

Plate No.	Time.	Neg. var.	Anel.	Katel.	0·001 volt.
	mins.				
2154	1	−5	..	..	10
	2	..	+ 9·5	..	
	3	..	..	−4	
	4	−3	..	..	
	5	..	+ 9·5	..	
	6	..	..	−3·5	
		Ether vapour.			
	7	−3·5	..	..	
	8	..	+ 7·5	..	
	9	..	..	−3·5	
	10	..	..	..	
	11	..	+ 4	..	
	12	..	..	−4·5	
	13	..	..	..	
	14	..	+ 7	..	
	15	..	..	−5	
	16	−4	..	..	
	17	..	+ 9	..	
	18	..	..	−4·5	
	19	−0·5	..	..	
	20	..	+11	..	
	21	..	..	−4	
	22	+2	..	..	
	23	..	+11	..	
	24	..	..	−4	8·5

Effect of Et_2O on katelectrotonus.

Plate No.	Time.	S.	0·001 volt.
	minutes.		
2157	1	7·5	
	5	7·5	
	6	Ether vapour for 1 min.	
	7	10·5	
	8	12	
	9	..	
	10	12·5	
	11	..	
	12	..	
	13	..	
	14	..	
	15	8	
	20	6	
	25	6	12·5

Effect of CO_2 upon anelectrotonus and katelectrotonus.

Plate No.	Time.	Anel. N.	Katel. S.	0·001 volt.
	minutes.			
2158	1	19·5	..	15
	2	..	3	
	9	19·5	..	
	10	..	3	
		CO_2 for 3 mins.		
	13	8	..	
	14	..	3	
	15	8	..	
	16	..	3·5	
	17	17·5	..	
	18	..	5	
	19	24·5	..	
	20	..	6·5	
	21	24·5	..	
	22	..	7	
	23	24	..	
	24	..	5	
	25	24	..	
	26	..	4·5	

Effect of Aconitine upon anelectrotonus and upon katelectrotonus.

Plate No.		Anel.	Katel.	0·001 volt.
2159	Before aconitine .	+15·0	−5·5	
	After aconitine .	+ 4·0	−4·0	
	After interpolar crush	+ 4·0	−4·0	

TABLE E (continued).—Data relating to Chapter IV., § 4.

Effect of Et_2O on katelectrotonus.

Plate No.	Time.	S.	0·001 volt.
2168	minutes.		
	1	12·5	13·5
	5	11·5	
	6	6·0	Ether vapour for 3 mins.
	7	3·0	
	8	3·0	
	9	3·0	
	10	3·5	
	11	3·5	
	12	4·0	
	13	4·0	
	14	4·5	
	15	5·0	
	20	6·5	
	25	7·0	12·5

Effect of Et_2O on anelectrotonus.

Plate No.	Time.	N.	0·001 volt.
2169 (fig. 68)	minutes.		
	1	10·5	13
	5	10·5	
	6	10·5	Ether vapour for 3 mins.
	7	4·0	
	8	3·0	
	9	2·0	
	10	2·5	
	11	3·0	
	12	3·0	
	13	3·5	
	14	4·0	
	15	4·0	
	20	8·5	
	25	11·5	
	30	12·5	12

Effect of Et_2O on anelectrotonus.

Plate No.	Time.	I. N. effect.	II. S. after-effect.	0·001 volt.
2170	minutes.			
	1	11·0	5·5	15
	5	11·0	5·0	
	6	10·5	Ether vapour for 4 mins.	
	7	4·0		
	8	2·5		
	9	2·0		
	10	2·0	0	
	11	3·0	0	
	12	3·5	0	
	13	3·5	0	
	14	3·5	0	
	15	4·0	0	
	20	6·0	0	
	25	8·5	0	
	30	10·5	0	14·5

Effect of $CHCl_3$ on anelectrotonus.

Plate No.	Time.	N.	0·001 volt.
2171	minutes.		
	1	11·0	14
	5	11·0	
	6	15·5	
	7	13·0	
	8	9·5	
	9	..	
	10	4·5	
	11	3·0	
	12	2·0	
	13	2·0	
	14	2·0	
	15	2·0	
	20	1·0	
	25	0·5	
	30	0·5	14

TABLE E (continued).—Data relating to Chapter IV., § 4.

Effect of CO_2 on neg. var. of katelectrotonus.

Plate No.	Time.		0·001 volt.
	minutes.		
2181	1	N. 15·5	20
(fig. 51)	5	„ 14·5	
	6	CO_2 for 1 min.	
	7	S. 6·0	
	8	„ 5·0	
	9	„ 4·5	
	10	„ 3·5	
	11	„ 3·5	
	12	N. 4·0	
	13	„ 6·0	
	14	„ 8·5	
	15	„ 10·0	
	20	„ 11·0	19

Effect of CO_2 on neg. var. of anelectrotonus.

Plate No.	Time.	S.	0·001 volt.
	minutes.		
2182	1	22	
	5	29	
	6	CO_2 for 1 min.	
	7	24	
	8	27	
	9	30	
	10	34	
	15	35	
	20	35	15

Effect of CO_2 on neg. var. of anelectrotonus.

Plate No.	Time.	S.	0·001 volt.
	minutes.		
2185	1	12·5	19
(fig. 52)	5	19·0	
	6	CO_2 for 1 min.	
	7	13·0	
	8	10·0	
	9	12·0	
	10	16·5	
	15	21·0	
	20	22·0	17·5

Effect of CO_2 on neg. var. of anelectrotonus.

Plate No.	Time.	S.	0·001 volt.
	minutes.		
2190	1	27·0	17
	5	27·0	
	6	CO_2 for 1 min.	
	7	24·5	
	8	28·0	
	9	30·0	
	10	31·0	
	11	30·0	
	12	29·5	
	13	27·5	
	14	26·5	
	15	25·0	
	16	24·0	
	17	23·5	
	18	23·0	

Effect of CO_2 on anelectrotonus.

Plate No.	Time.	N.	0·001 volt.
	minutes.		
2192	1	43·5	16·5
	5	44	
	6	CO_2 for 1 min.	
	7	30	
	8	35	
	9	39	
	10	Off plate	
	15	55	
	20	57	

Effect of CO_2 on anelectrotonus.

Plate No.	Time.	N.	0·001 volt.
	minutes.		
2194	1	22	22
	5	25	
	6	CO_2 for 1 min.	
	7	14	
	8	17	
	9	19	
	10	Off plate	
	15	„	
	20	37·5	21

TABLE E (continued).—Data relating to Chapter IV., § 4.

Effect of CO_2 on katelectrotonus.

Plate No.	Time.	S.	0·001 volt.
	minutes.		
2195	1	12	19
(fig. 49)	5	12·5	
	6	CO_2 for 1 min.	
	7	17·5	
	8	16·5	
	9	18·5	
	10	23·0	
	11	26·5	
	12	28·5	
	13	27·0	
	14	25·0	
	15	23·0	
	20	18·0	
	25	15·0	19

Effect of CO_2 on anelectrotonus.

Plate No.	Time.	N.	0·001 volt.
	minutes.		
2196	1	26 0	
(fig. 50)	5	26·0	
	6	CO_2 for 1 min.	
	7	15·0	
	8	20·0	
	9	22·0	
	10	24·0	
	15	38·5	
	20	36·5	
	25	33·0	18

Effect of CO_2 on katelectrotonus.

Plate No.	Time.	S.	0·001 volt.
	minutes.		
2197	1	11·5	18·5
	5	11·5	
	6	CO_2 for 1 min.	
	7	13·5	
	8	14·0	
	9	16·5	
	10	21·0	
	11	26·0	
	12	27·0	
	13	25·0	
	14	22·5	
	15	20·5	
	20	16·5	18

Effect of CO_2 on anelectrotonus.

Plate No.	Time.	N.	0·001 volt.
	minutes.		
2198	1	29	24
	5	30	
	6	CO_2 for 1 min.	
	7	19	
	8	22	
	9	24	
	10	27	
	15	Off plate	
	20	36	

Effect of CO_2 on anelectrotonus.

Plate No.	Time.	N.	0·001 volt.
	minutes.		
2199	0	6·5	7·5
	5	7·0	
	6	10·0	CO_2
	7	13·5	
	8	14·0	
	9	14·5	
	10	15·0	
	15	12·5	
	20	9·5	
	25	8·5	
	30	7·5	7·0

Effect of CO_2 on anelectrotonus.

Plate No.	Time.	N.	0·001 volt.
	minutes.		
2200	1	21·0	
	5	21·0	
	6	CO_2 for 1 min.	
	7	9·5	
	8	11·5	
	9	13·0	
	10	15·0	
	11	17·0	
	12	19·0	
	13	21·0	
	14	22·0	
	15	23·0	

TABLE E (continued).—Data relating to Chapter IV., § 4.

Effect of aconitine on neg. var., on anel., and on katel.

Plate No.	Time.	Neg. var.	Anel.	Katel.	0·001 volt.
	mins.				
2201	1	−9	..	..	11·5
	2	..	+14	..	
	3	..	..	−4·5	
	4	−8	..	..	
	5	..	+14	..	
	6	..	..	−4·0	
	7	−7	..	..	
	8	..	+14	..	
	9	..	..	−4·0	
	Bath of Aconitine Hydrochloride 1 per cent. for 1 minute.				
	14	−5·5	..	..	11·0
	15	..	+ 9·0	..	
	16	..	..	−2·5	
	17	−4.5	..	..	
	18	..	+ 5·5	..	
	19	..	..	−2·5	
	20	−0·5	..	..	
	21	..	+ 4·0	..	
	22	..	..	−2·5	
	23	0·0	..	..	
	24	..	+ 2·5	..	
	25	..	..	−2·5	11·5
	26	Nerve crushed in interpolar region			
	27	0·0	..	..	
	28	..	0·0	..	
	29	..	..	0·0	11

Effect of CO_2 on N. polarisation increment.

Plate No.	Time.	N.	0·001 volt.
	minutes.		
2203 (fig. 56)	1	17·5	13·5
	5	18·5	
	6	CO_2 for 1 min.	
	7	4·5	
	8	9·0	
	9	7·5	
	10	5·0	
	11	5·5	
	12	9·5	
	13	14·0	
	14	16·5	
	15	18·5	
	20	22·5	
	25	23·5	13·5

Effect of CO_2 on S. polarisation increment.

Plate No.	Time.	S.	0·001 volt.
	minutes.		
2204 (fig. 55)	1	13·0	12
	5	13·0	
	6	CO_2 for 1 min.	
	7	12·0	
	8	8·0	
	9	9·0	
	10	11·5	
	11	15·5	
	12	19·5	
	13	21·5	
	14	23·5	
	15	23·5	
	20	20·5	
	25	18·5	12·5

Effect of CO_2 on S. polarisation increment.

Plate No.	Time.	S.	0·001 volt.
	minutes.		
2205	1	12·0	12
	5	12·0	
	6	CO_2 for 1 min.	
	7	8·0	
	8	7·0	
	9	..	
	10	10·5	
	15	18·5	
	20	17·0	11·5

Effect of CO_2 on N. polarisation increment.

Plate No.	Time.	N.	0·001 volt.
	minutes.		
2206	1	17·0	12·5
	5	16·5	
	6	CO_2 for 1 min.	
	7	..	
	8	5·0	
	9	6·5	
	10	8·0	
	15	17·5	
	20	18·5	12·5

TABLE E (continued).—Data relating to Chapter IV., § 4.

Effect of CO_2 on polarising current.

Plate No.	Time.	S.	0·001 volt.
	minutes.		
2207	1	19·5	17
(fig. 53)	5	19·5	
	6	CO_2 for 2 min.	
	7	19·5	
	8	19·5	
	9	19·5	
	10	19·5	
	15	19·5	
	20	19·5	17
		N.	
2208	1	19·5	17
(fig. 54)	5	19.5	
	6	CO_2 for 2 min.	
	7	19·5	
	8	19·5	
	9	19·5	
	10	19·5	
	15	19·5	
	20	19·5	17

Effect of Et_2O on neg. var. of anelectrotonus.

Plate No.	Time.	S.	0·001 volt.
	minutes.		
2209	1	33	13
	5	33	
	6	39 }	Ether vapour
	7	24 }	for 2 mins.
	8	11	
	9	11	
	10	9	
	11	13	
	12	17	
	13	22	
	14	26	
	15	30	
	16	32	
	17	33	
	18	34	
	19	34	13

Effect of $CHCl_3$ on neg. var., on anelectrotonus, and on katelectrotonus.

Plate No.	Time.	Neg. var.	Anel.	Katel.	0·001 volt.
	mins.				
2211	1	−8	..	..	12
	2	..	+11	..	
	3	..	..	−5	
	4	−7	..	..	
	5	..	+ 9	..	
	6	..	..	−4·5	
	7	−7	..	..	
	8	..	+10	..	
	9	..	..	−5	
	10 }	Chloroform vapour			
	11 }				
	15	0	..	..	
	16	..	+ 1	..	
	17	..	..	−1	
	30	0	..	..	
	31	..	+ 1	..	
	32	..	..	−1	

Effect of Et_2O on katelectrotonus.

Plate No.	Time.	S.	0·001 volt.
	minutes.		
2220	1	24·0	9
	5	24·5	
	6	27·0 }	Ether vapour
	7	30·5 }	for 2 mins.
	8	29·5	
	9	33·0	
	10	34·5	
	11	36·0	
	12	37·0	
	13	37·5	
	14	37·0	
	15	36·0	
	20	31·0	
	25	26·5	

TABLE E (continued).—Data relating to Chapter IV., § 4.

Effect of Et_2O on anelectrotonus.

Plate No.	Time.	N.	0·001 volt.
	minutes.		
2221	1	52·0	10
	5	54·0	
	6	.. }	Ether vapour
	7	42·0 }	for 2 mins.
	8	41·0	
	9	42·0	
	10	44·0	
	11	45·5	
	12	47·0	
	13	48·0	
	14	49·5	
	15	50·0	
	20	51·0	
	25	51·5	10

Effect of Et_2O on S. polarisation increment.

Plate No.	Time.	S.	0·001 volt.
	minutes.		
2223	1	27·0	12·5
	5	27·0	
	6	.. }	Ether vapour
	7	.. }	for 2 mins.
	8	..	
	9	0·0	
	10	0·0	
	11	1·0	
	12	6·5	
	13	18·0	
	14	..	
	15	24·5	
	20	23·0	12

Effect of Et_2O on N. polarisation increment.

Plate No.	Time.		0·001 volt.
	minutes.		
2224	1	N. 20·0	
	5	„ 18·5	
	6	„ .. {	Ether vapour
	7	„ .. {	for 2 mins.
	8	„ ..	
	9	„ 0·0	
	10	„ 0·0	
	11	S. 1·0	
	12	„ 3·0	
	13	„ 10·0	
	14	„ 11·0	
	15	„ 6·0	
	16	N. 1·5	
	17	„ 4·5	
	18	„ 7·0	
	19	„ 8·0	
	20	„ 10·0	
	21	„ 11·0	
	22	„ 11·5	11

Effect of Et_2O on S. polarisation increment.

Plate No.	Time.	S.	0·001 volt.
	minutes.		
2225	1	12·0	13
	5	12	
	6	.. {	Ether vapour
	7	.. {	for 2 mins.
	8	0·0	
	9	0·0	
	10	0·0	
	11	1·0	
	12	1·0	
	13	3·0	
	14	11·0	
	15	15·5	
	20	16·0	13

TABLE E (continued).—Data relating to Chapter IV., § 4.

Effect of $CHCl_3$ on S. polarisation increment.

Plate No.	Time.	S.	0·001 volt.
	minutes.		
2226	1	13·0	11·5
	5	13·0	
	6	6·0	Chloroform vapour for 2 mins.
	7	2·0	
	8	0·0	
	9	0·0	
	10	0·0	
	11	0·0	
	12	0·0	
	13	0·0	
	14	0·0	
	15	0·5	
	20	1·5	

Effect of Et_2O on N. polarisation increment.

Plate No.	Time.		0·001 volt.
	minutes.		
2227	1	N. 14·0	14
	2	,, 14·0	
	3	,, 14·0	
	4	,, 14·0	
	5	,, 14·0	
	6	,, ..	Ether vapour for 2 mins.
	7	,, ..	
	8	,, ..	
	9	S. 2·5	
	10	,, 2·5	
	11	,, 1·5	
	12	N. 6·0	
	13	,, 9·5	
	14	,, 12·0	
	15	,, 12·5	
	20	,, 13·5	13·5

Effect of $CHCl_3$ on N. polarisation increment.

Plate No.	Time.	N.	0·001 volt.
	minutes.		
2228	1	14·5	13·5
	5	14·5	
	6	0·0	Chloroform vapour for 2 mins.
	7	0·0	
	10	0·0	
	15	0·0	
	20	0·0	13·5

Effect of $CHCl_3$ on anelectrotonus.

Plate No.	Time.	N.	0·001 volt.
	minutes.		
2231	1	24·5	15
	5	25·0	
	6	24·0	Chloroform vapour for 2 mins.
	7	16·0	
	8	9·5	
	9	8·5	
	10	9·0	
	15	6·0	
	20	5·5	
	25	5·5	15

Effect of $CHCl_3$ on katelectrotonus.

Plate No.	Time.	S.	0·001 volt.
	minutes.		
2232	1	6·5	6·5
	5	6·5	
	6	..	Chloroform vapour for 2 mins.
	7	6·0	
	8	6·0	
	9	5·0	
	10	4·5	
	15	2·5	
	20	1·5	
	25	1·5	7

TABLE E (continued).—Data relating to Chapter IV., § 4.

Effect of Et_2O on katelectrotonus.

Plate No.	Time.	S.	0·000 volt.
	minutes.		
2233	0	10·5	11
	5	10·5	
	6	11·0	
	7	10·0	Et_2O
	8	..	
	9	9·0	
	15	7·0	
	20	6·5	
	25	5·5	
	30	5·0	11

Effect of CO_2 on neg. var., on anelectrotonus, and on katelectrotonus.

Plate No.	Time.	Neg. var.	Anel.	Katel.	0·001 volt.
	mins.				
2235	1	− 3·5	..	..	9·5
	2	..	+ 7·5	..	
	3	..	..	−1·5	
		CO_2 for 1 min.			
	5	7·5	..	..	
	6	..	2·5	..	
	7	..	..	1·5	
	8	10·5	..	..	
	9	..	7·0	..	
	10	..	..	7·0	
	11	4	..	..	
	12	..	18·0	..	
	13	..	..	2·5	
	14	1·5	..	..	
	15	..	14·5	..	
	16	..	..	4·0	
	17	2·5	..	..	
	18	..	9·5	..	
	19	..	..	4·5	
	20	2	..	..	
	21	..	+ 8	..	
	22	..	..	−4	
	23	− 2	..	..	
	24	..	+ 7	..	
	25	..	..	−4	

Effect of CO_2 on neg. var., on anelectrotonus and on katelectrotonus.

Plate No.		Neg. var.	Anel.	Katel.	0·001 volt.
2236	Before CO_2 .	−4	+9·5	−4·5	10
	10 minutes after CO_2	−9	+12	−7·5	

All deflections abolished by crushing the nerve between exciting and leading-off electrodes.

Effect of $CHCl_3$ on neg. var. of katelectrotonus.

Plate No.	Time.	N.	0·001 volt.
	minutes.		
2237	1	20	13·5
	5	18	
	6	0	Chloroform vapour for 2 min.
	7	0	
	8	0	
	9	0	
	10	0	
	15	0	
	20	0	
	25	0	14·5

Effect of $CHCl_3$ on anelectrotonus.

Plate No.	Time.	N.	0·001 volt.
	minutes.		
2239	0	9·5	9·0
	5	9·5	
	6	9·0	
	7	3·5	
	8	..	
	9	3·5	
	10	..	
	11	3·5	
	16	3·5	
	21	3·5	
	29	3·5	10·5

This deflection persisted after the nerve had been completely crushed between the exciting and leading-off electrodes.

TABLE E (continued).—Data relating to Chapter IV., § 4.

Effect of Et_2O on anelectrotonus and katelectrotonus.

Plate No.	Time.	Anel.	Katel.	0·001 volt.
		minutes.		
2288-9	1	+8·5	..	10
	2	..	−2·0	
	3	+8·5	..	
	4	..	−2·5	
	5	+8·5	..	
	6	..	−2·5	
	7	+9·0	..	
	8	..	..	
		Ether vapour for 2 mins.		
	15	+1·5	..	
	16	..	−1·5	
	25	+1·0	..	
	26	..	−0·5	
	33	+1·5	..	
	34	..	−0·5	10
		Pause of 30 mins.		
	64	+5 5	..	9
	65	..	−2·5	
	66	+5·5	..	
	67	..	−2·5	
	68	+5·5	..	
	69	..	−2·5	
		Ether vapour for 2 mins.		
	72	+trace	..	
	73	..	−trace	
	78	+trace	..	
	79	..	−trace	
		Nerve crushed in interpolar region.		
	81	+trace	..	
	82	..	−trace	

Effect of ether on neg. var., on anel., and on katel.

Plate No.	Time.	Neg. var.	Anel.	Katel.	0·001 volt.
	mins.				
2290	1	−10·5	..	..	12
	2	..	+10	..	
	3	..	..	−1·5	
	4	−10	..	..	
	5	..	+ 9	..	
	6	..	..	−1·5	
	7	−10	..	..	
	8	..	..	..	
	9	..	..	−1·5	
		Ether vapour for 3 minutes			
	13	0·0	..	..	
	14	..	+ 4·5	..	
	15	..	..	−1	
	16	0·0	..	..	
	17	..	+ 4·5	..	
	18	..	..	−1	
	19	0·0	..	..	
	20	..	+ 3·5	..	
	21	..	..	−1	
	22	0·0	..	..	
	23	..	+ 3	..	
	24	..	..	−1	
	25	0·0	..	..	
	26	..	+ 2	..	
	27	..	..	−0·5	12·5
		Nerve crushed in interpolar region			
	31	0·0	..	..	
	32	..	0·0	..	
	33	..	..	0·0	12

TABLE E (continued).—Data relating to Chapter IV., § 4.

Effect of Et_2O on anelectrotonus and katelectrotonus.

Plate No.	Time.	Anel.	Katel.	0·001 volt.
	minutes.			
2291	1	+31	..	12·5
	2	..	−6	
	3	+31	..	
	4	..	−5·5	
	5	+31	..	
	6	..	−6	
	7	+29·5	..	
	8	..	−5·5	
	9	+29·5	..	
	10	..	−5·5	
		Ether vapour		
	11	+ 9·5	..	
	12	..	−6	
	13	+ 5	..	
	14	..	−5	
	15	+ 6	..	
	16	..	−4·5	
	17	+ 6	..	
	18	..	−4	
	19	+ 5	..	
	20	..	−3	
	21	+ 5	..	
	22	..	−3	
	23	+ 4	..	
	24	..	−2	
	25	+ 4	..	12·5
		Nerve crushed in interpolar region		
	27	..	− trace	
	28	+ trace	..	
	29	..	− trace	
	30	+ trace	..	12

Effect of ether on anelectrotonus.

Plate No.	Time.		0·001 volt.
	minutes.		
2292	1	+35	13
	5	+35	
	10	+36	
	11	+35	
	12	+18	
	13	+10·5	Ether vapour
	14	+10·5	
	15	+ 8	
	16	+ 7	
	17	+ 6·5	
	18	+ 6	
	19	+ 6	
	20	+ 6	
	25	+ 4·5	
	30	+ 4	
	35	+ 3	
	40	+ 3	
	45	+ 3	12·5

No deflection after interpolar crush.

Effect of ether on neg. var. and on anelectrotonus.

Plate No.	Time.	Neg. var.	Anel.	0·001 volt.
	mins.			
2293	1	−6·5	..	14·0
	2	..	+17·0	
	9	−7·5	..	
	10	..	+18·0	
		Ether vapour for 4 minutes.		
	15	0·0	..	
	16	..	+ 6·0	
	23	0·0	..	
	24	..	+ 4·0	
	29	0·0	..	
	30	..	+ 3·5	
	35	0·0	..	
	36	..	+ 4·0	
	41	0·0	..	
	42	..	+ 4·5	12·5

No deflection after interpolar crush.

TABLE E (continued).—Data relating to Chapter IV., § 3.

Effect of tetanisation upon katelectrotonus.

Plate No.	Time.		0·001 volt.
	minutes.		
2294	0	+8·5	12·5
	2	−8·5	
	4	−8·5	
	6	−8·5	
		Tetanisation, 30 units, 5 mins.	
	14	−9·5	
	16	−9·0	
	18	−9·0	
	20	−9·0	
	22	−9·0	
	24	−9·0	
	26	−9·0	

Effect of tetanisation upon anelectrotonus.

Plate No.	Time.		0·001 volt.
	minutes.		
2295	0	+30·5	
	2	+30·5	
	4	+30·0	
	6	+31·5	
		Tetanisation, 50 units, 5 mins.	
	13	+34·5	
	15	+34·5	
	17	+34·0	
	19	+33·0	
	21	+32·5	
	23	+32·0	

Effect of tetanisation upon anelectrotonus.

Plate No.	Time.		0·001 volt.
	minutes.		
2296	0	+18·0	9
	5	+19·0	
	10	+19·0	
		Tetanisation, 50 units, 5 mins.	
	18	+12·0	
	19	+12·5	
	20	+13·0	
	25	+14·0	
	30	+14·5	
		Nerve crushed in interpolar region.	
	32	+ 2·5	

F.—Time Table.

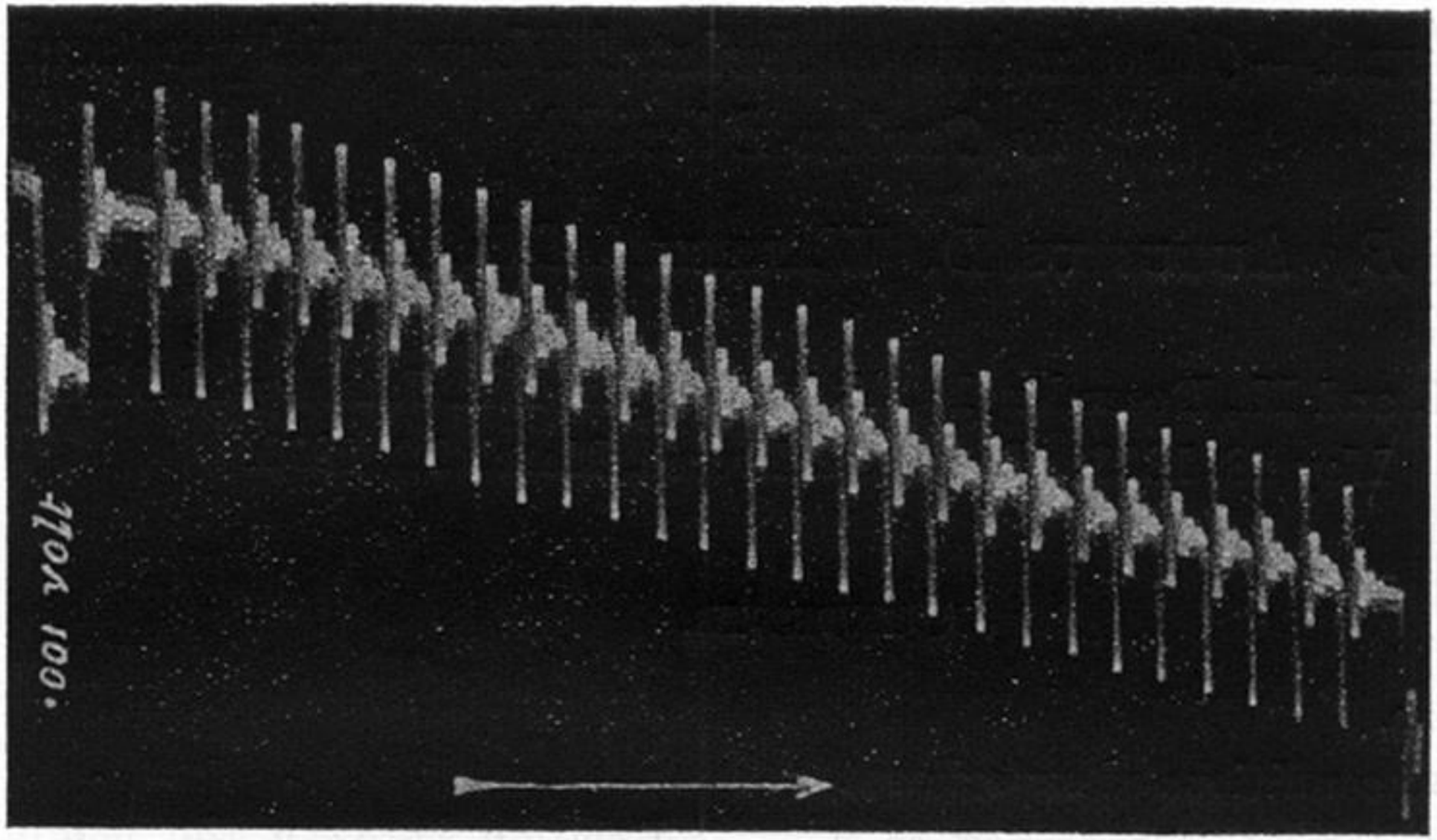
In view of possible differences in nerve-reaction with season, the following Time Table is added, by reference to which the date of any given observation may be approximately determined.

Plate No.	1,	January	3,	1895.
,, ,,	50,	February	1,	,,
,, ,,	100,	,,	25,	,,
,, ,,	150,	March	6,	,,
,, ,,	200,	July	6,	,,
,, ,,	250,	,,	16,	,,
,, ,,	300,	,,	25,	,,
,, ,,	350,	August	4,	,,
,, ,,	400,	,,	12,	,,
,, ,,	450,	,,	20,	,,
,, ,,	500,	,,	26,	,,
,, ,,	550,	October	5,	,,
,, ,,	600,	,,	13,	,,
,, ,,	650,	,,	22,	,,
,, ,,	700,	November	2,	,,
,, ,,	750,	,,	9,	,,
,, ,,	800,	,,	21,	,,
Plate No.	850,	November	28,	1895.
,, ,,	900,	December	5,	,,
,, ,,	950,	,,	13,	,,
,, ,,	1000,	January	3,	1896.
,, ,,	2000,	,,	,,	,,
,, ,,	2050,	,,	10,	,,
,, ,,	2100,	,,	21,	,,
,, ,,	2150,	February	3,	,,
,, ,,	2200,	,,	13,	,,
,, ,,	2250,	March	6,	,,
,, ,,	2250 to 2300,	July,		,,
,, ,,	3000,	May	10,	,,
,, ,,	3050,	June	2,	,,
,, ,,	3100,	,,	16,	,,
,, ,,	3150,	July	1,	,,
,, ,,	3200,	,,	18,	,,

Note.—Several points referred to in the lecture as delivered, in order to justify the extended and systematic application of the method to chemico-physiological questions, and mentioned therefore in the lecture-abstract ('Roy. Soc. Proc.,' March 12, 1896), have not been discussed in the foregoing paper, the principal theme of which is constituted by the discussion of the relations of carbon dioxide to the electrical reactions of medullated nerve. These points (action of anæsthetics, of acids, bases and salts, of alkaloids and narcotics, of aconitine and its constituents, &c.) will be presented *in extenso* in further papers. The action of anæsthetics is considered in some detail in the current number of 'Brain,' June, 1896.)

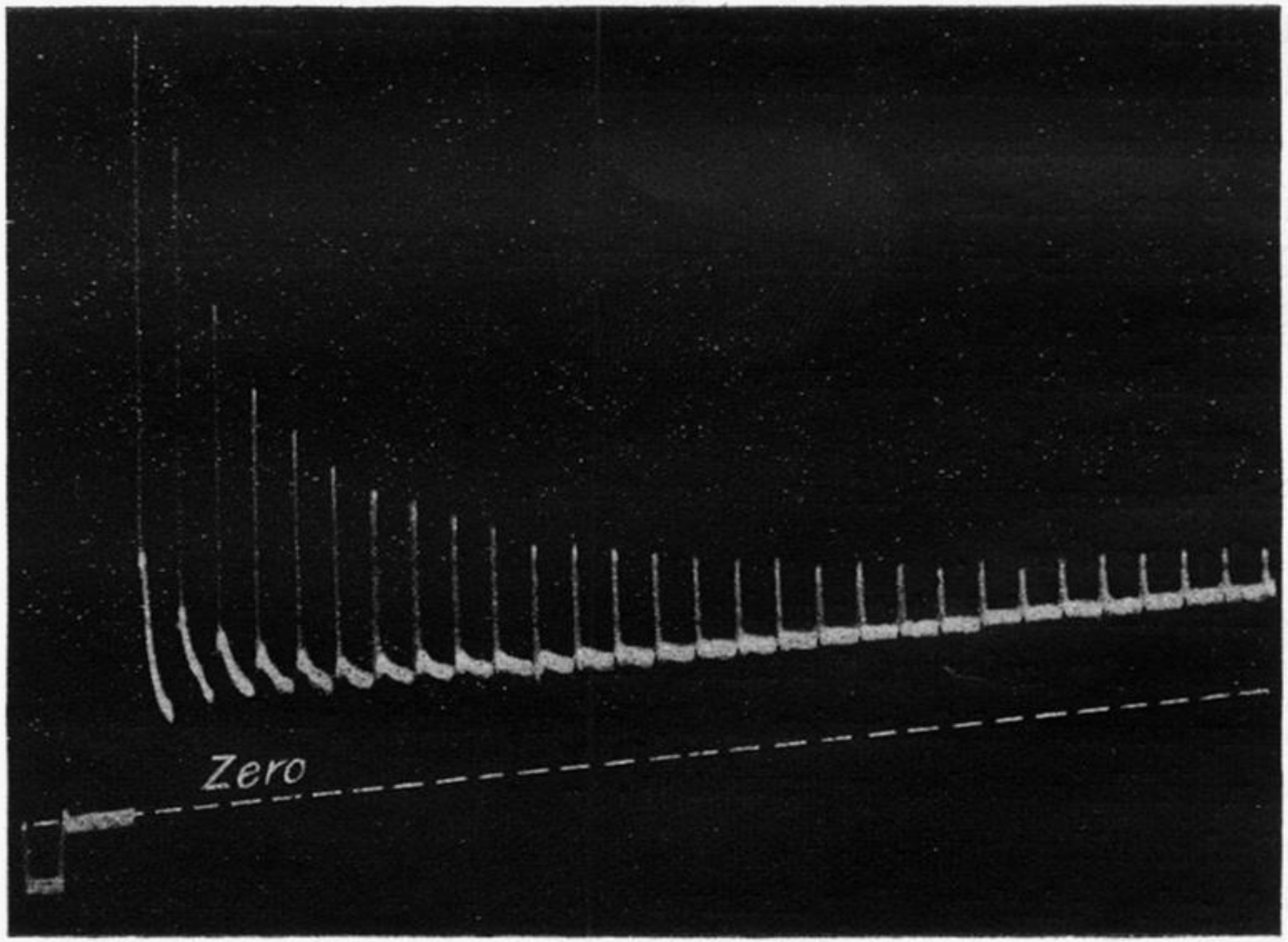
I desire to make some slight acknowledgment of the help that I have received from Miss S. C. M. Sowton, in carrying out experiments. The undivided attention and great neatness with which Miss Sowton has worked have furnished me with a much larger body of useful data than I could otherwise have obtained. Many of the most successful records have been obtained for me in my absence.

Fig. 1. (Obs. 334.



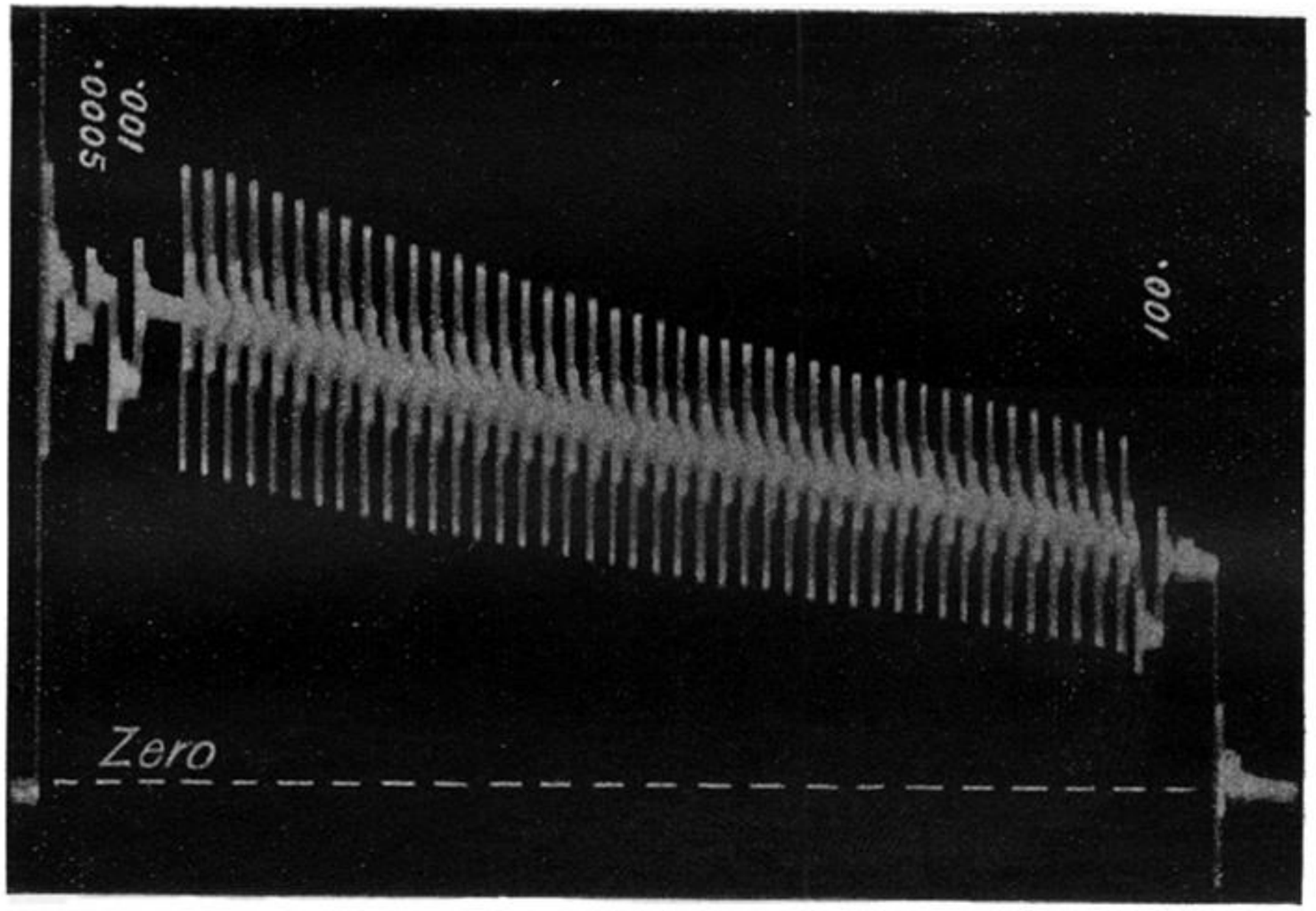
Normal series of negative variations of current of injury of nerve. Excitation each minute for $\frac{1}{8}$ minute. The general decline south signifies a declining current of injury. Each negative deflection consists of a southward swing, followed by instrumental oscillations above and below the position of rest of the suspended magnets of a Thomson galvanometer. The deflection at the beginning of the series is by 0·001 volt.

Fig. 2. (Obs. 1501.)



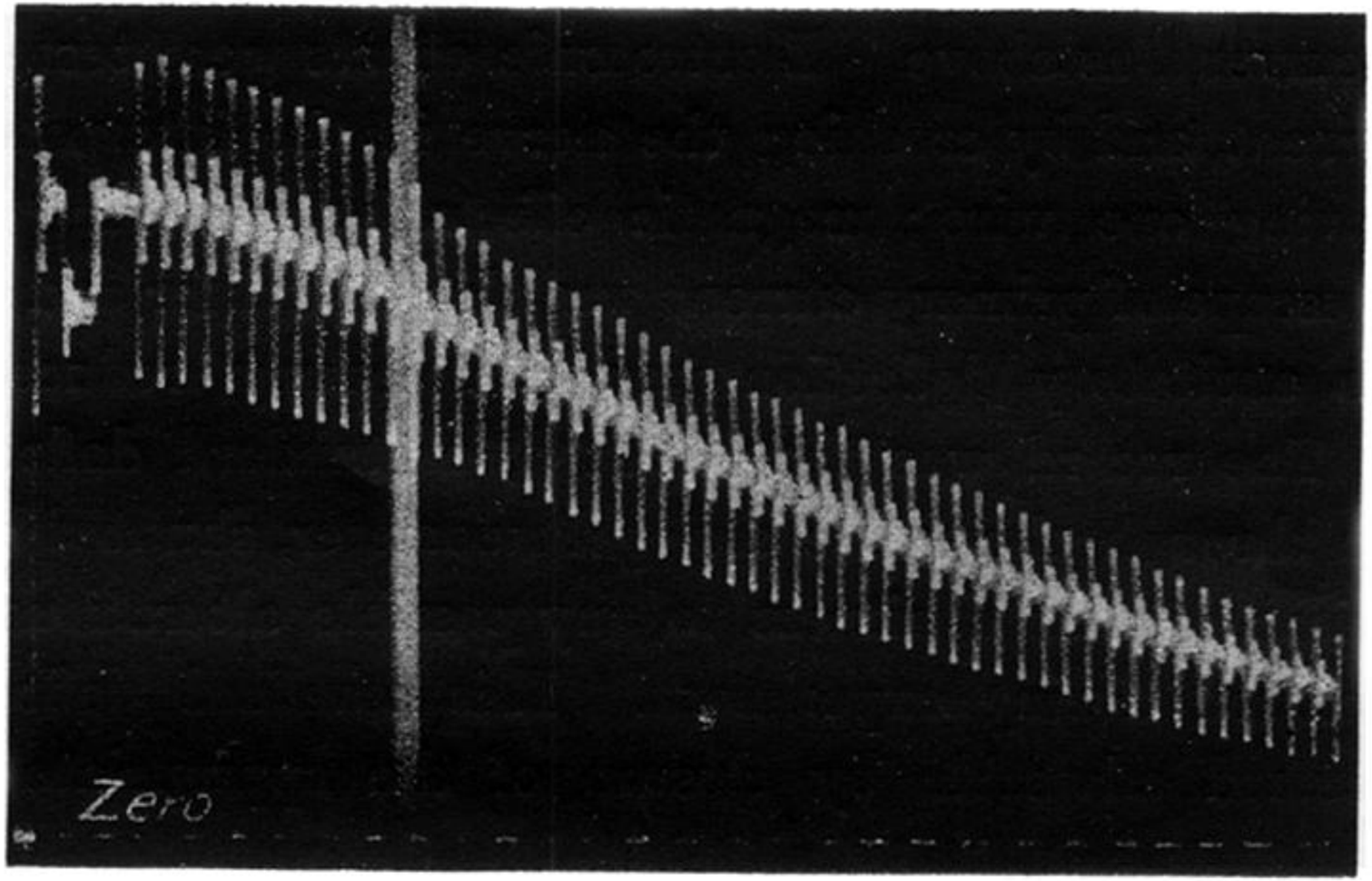
Series of negative variations of current of injury of muscle, to show the contrast between their rapid decline and the undiminishing, or slightly diminishing, series of negative variations of nerve. Maximal indirect tetanisation for 7·5 seconds, at intervals of 1 minute. The deflection at commencement of record is by 0·001 volt. The rise of the base line is accidental. Dead-beat galvanometer.

Fig. 4. (Obs. 309.)



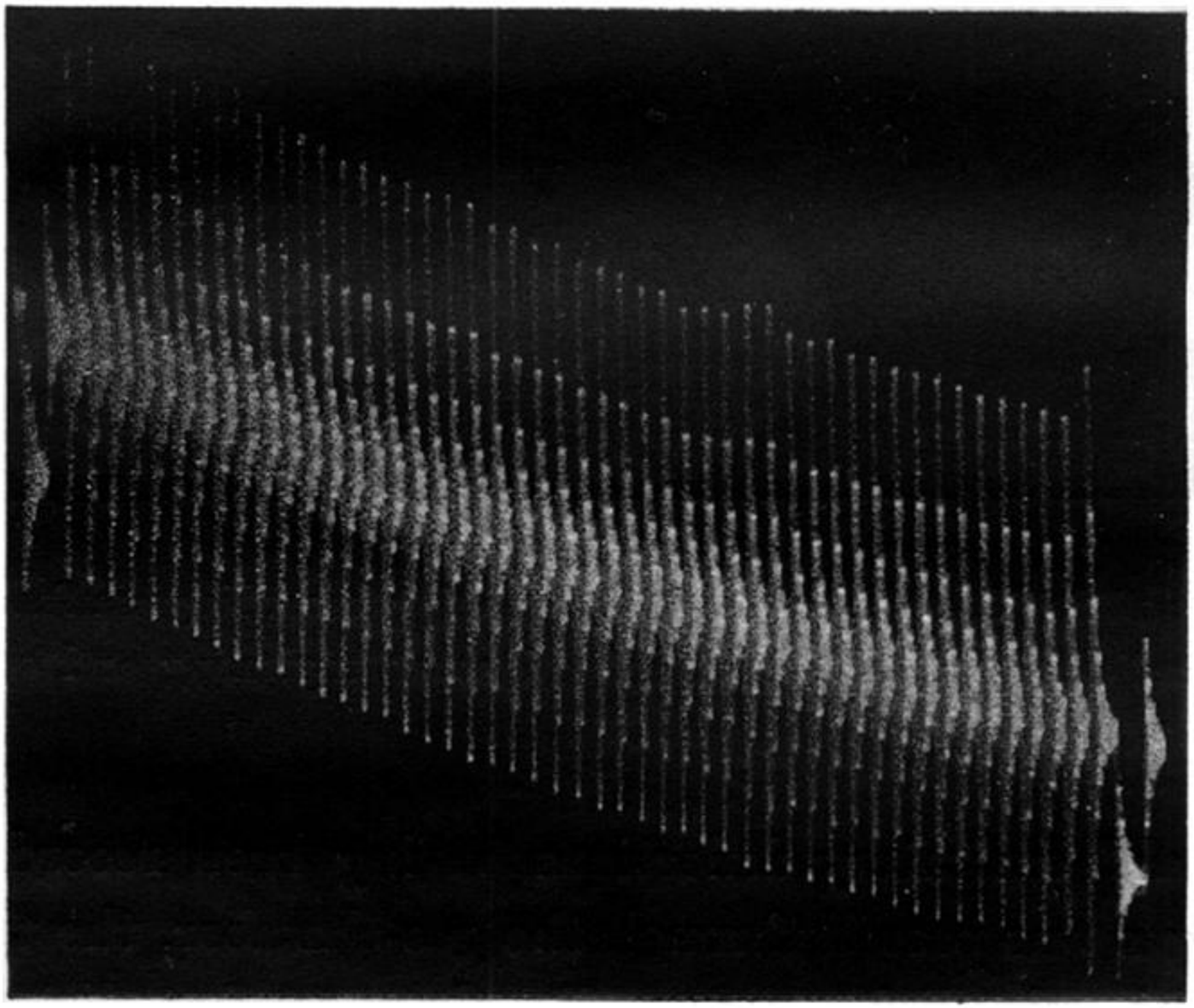
Similar series to that of fig. 1, on a more slowly moving recording surface. The zero line of the galvanometer is indicated; the standard deflections at beginning and end of the record are by $\frac{1}{1000}$ volt.

Fig. 5. (Obs. 336.)



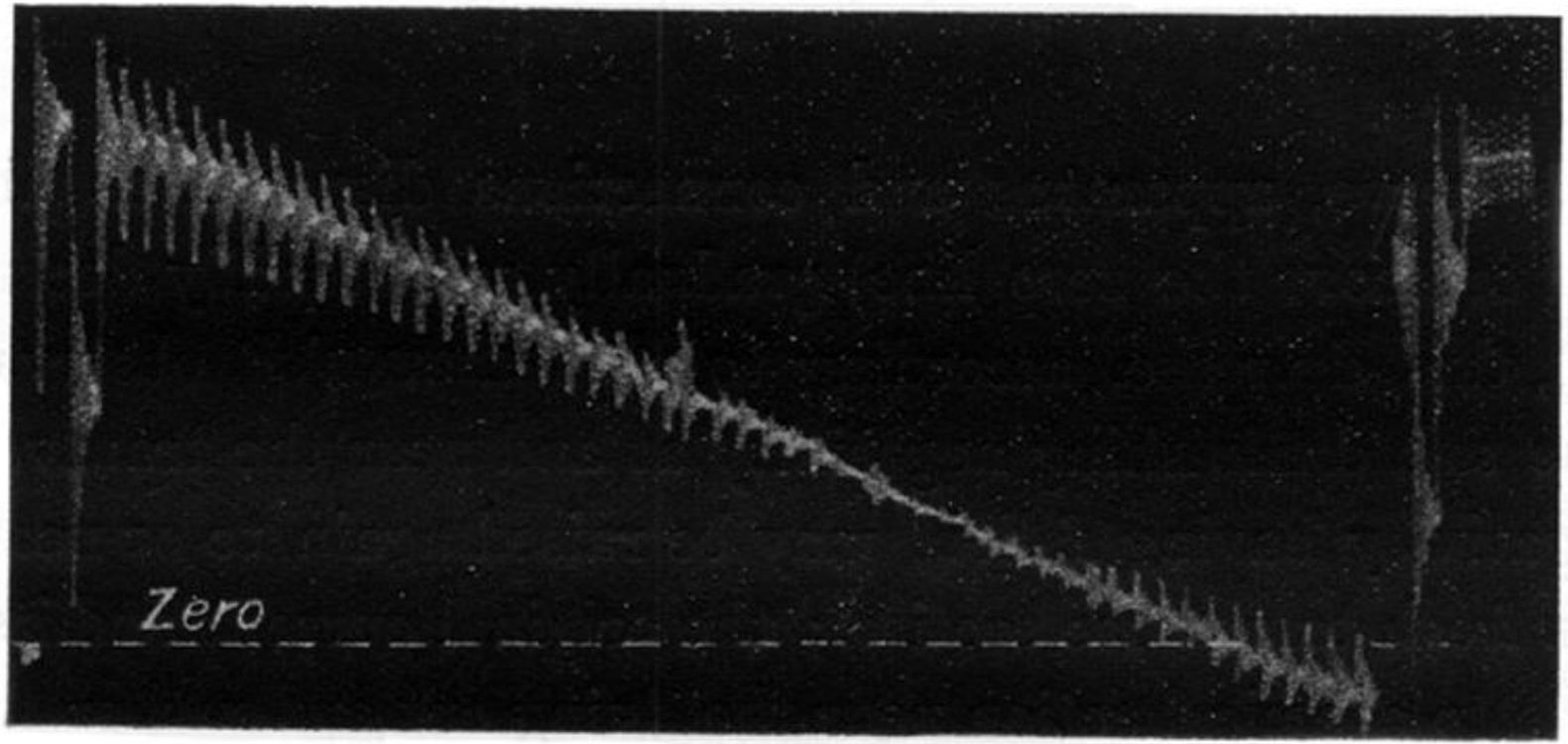
Similar series. Negative variation declining more rapidly than usual. Nerve left for about an hour in the pithed frog before use. The vertical white bar marks where nitrous oxide was passed into the nerve-chambers.

Fig. 6. (Obs. 708.)



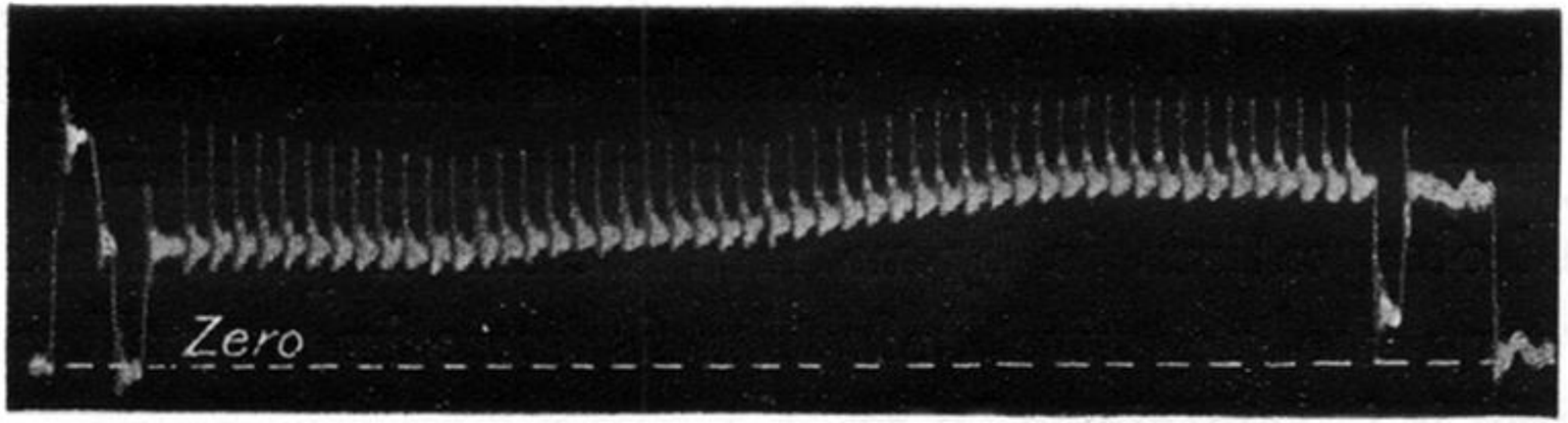
Similar series. Negative variation of nearly constant magnitude. The excised nerve had been kept for 18 hours in physiological saline. The difference in the decline of the negative variation in this and in preceding figure is probably due to the action of CO_2 in the case of fig. 5, and the absence of such action in the case of fig. 6. The positive after-effect is well marked, giving an upward excursion of the magnet considerably in excess of the instrumental oscillation. (See pp. 66 and 84.)

Fig. 7. (Obs. 279.)



Gradual reversal of the "normal" (south or negative) deflection, and appearance of a reversed (north or positive) deflection.

Fig. 8. (Obs. 842.)



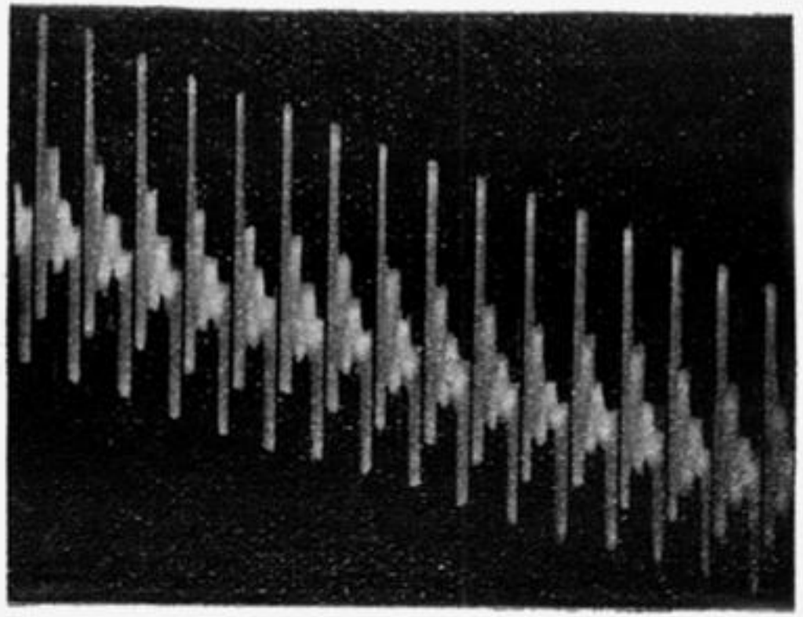
Reversed or positive variation.

The nerve had been kept in physiological saline for $8\frac{1}{2}$ hours after removal, and had been used the hour previously for an experiment with CO_2.

When first observed the variation was composed of a small negative deflection followed by a large positive after-deflection. The variation in the figure is composed of a positive deflection (during excitation) followed by a second positive deflection (at the end of excitation).

The period of the magnet is 15 seconds; the relation of its first swing from the position of rest to its first swing-back beyond that position is $\frac{+36}{-17}$. The duration of excitation is $7\frac{1}{2}$ seconds.

Fig. 9. (Obs. 102.)



Portion of a series to show state of nerve intermediate between the first state (predominant south or negative excitatory deflection), and the third state (predominant north or positive excitatory deflection), viz., in which a small negative effect is followed by a larger positive after-effect.

The oscillation decrement in conjunction with the duration of excitation is such that a normal swing-back is equal to two-thirds of the original swing. In this case, therefore, a negative deflection of 7·5 has a positive oscillation of 5 millims., and the net positive after-effect is obtained by subtracting this value from the recorded after-effect, *i.e.*, by subtracting 5 from 12.

Fig. 10. (Obs. 101.)

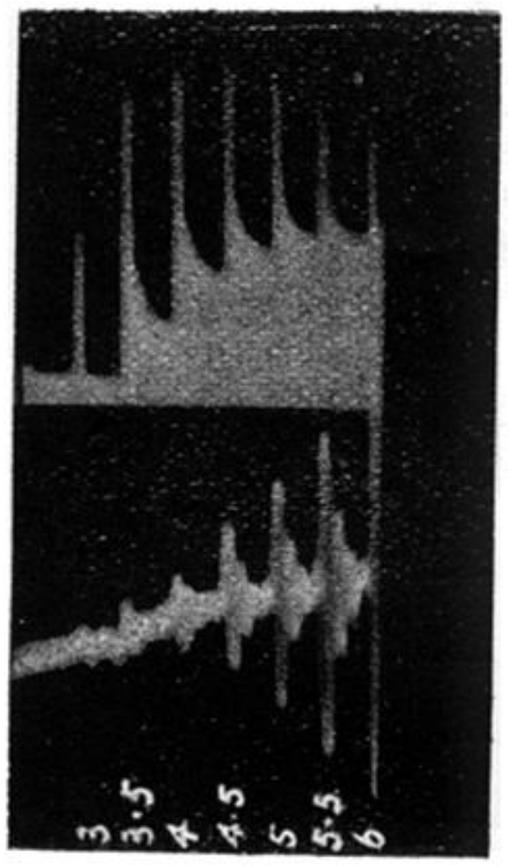


Fig. 11. (Obs. 134.)

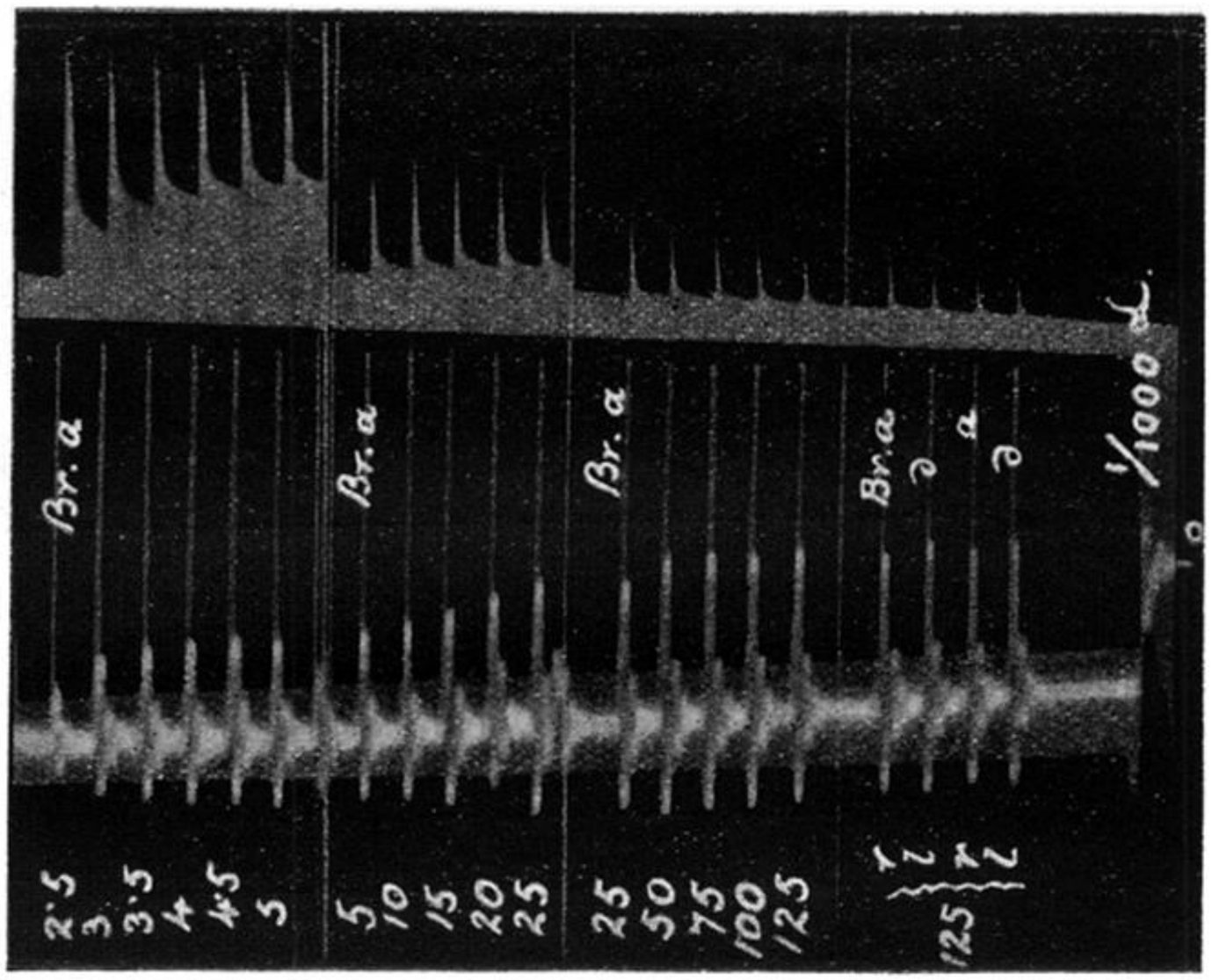


Fig. 10. Simultaneous record of the negative variation of nerve and of the contraction of muscle with increasing strength of stimulation (2·5 to 125 units of Berne coil).

The muscular response is dying off during the experiment, by lapse of time and by fatigue. The nerve response is kept up at a normal level, and increases in magnitude with increasing strength of stimulation (2·5 to 5 units of Berne coil).

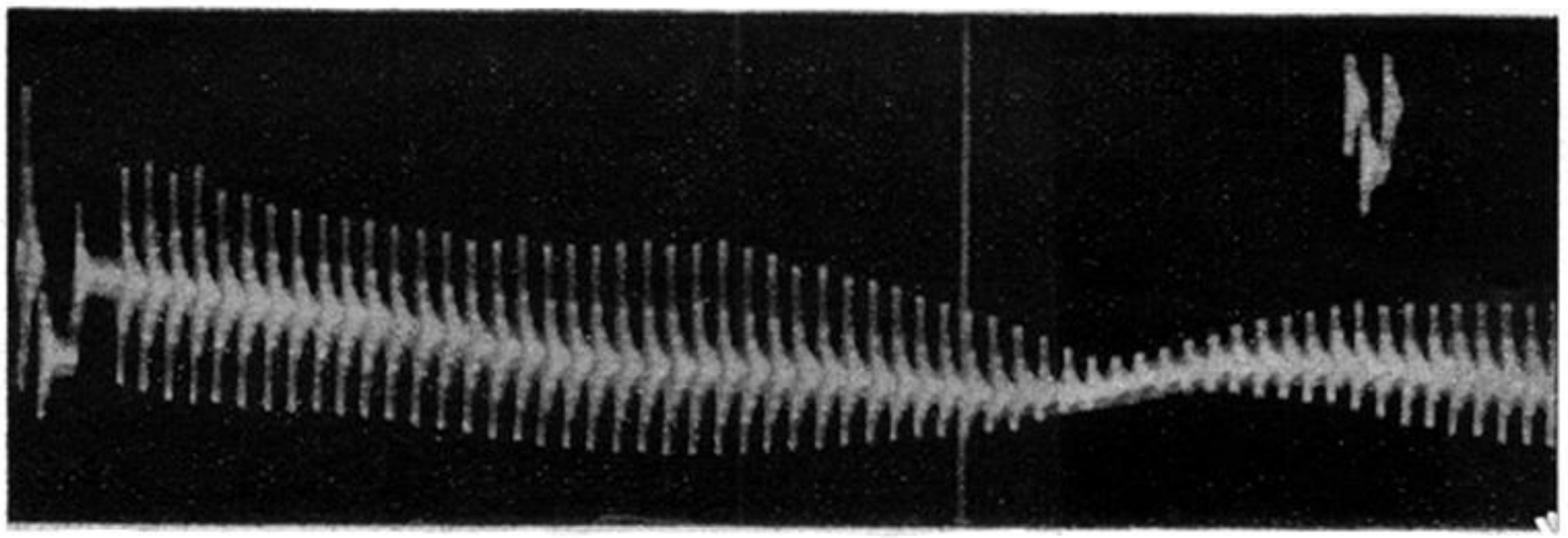
The muscular response is dying off during the experiment, by lapse of time and by fatigue. The nerve response is kept up at a normal level, and increases in magnitude with increasing strength of stimulation, from 2·5 to 5, from 5 to 25, and more slowly up to about 50 units; at 125 units tests are made with reversal of direction of exciting currents, their inequality indicates that at this strength unipolar or electrotonic effects may become sensible; at lower strengths reversal of the exciting current was of no effect upon the magnitude of deflection; at the lowest, viz., minimal strengths differences were obtained in the nerve as well as in the muscular response signifying differences in excitation according to its direction (break currents).

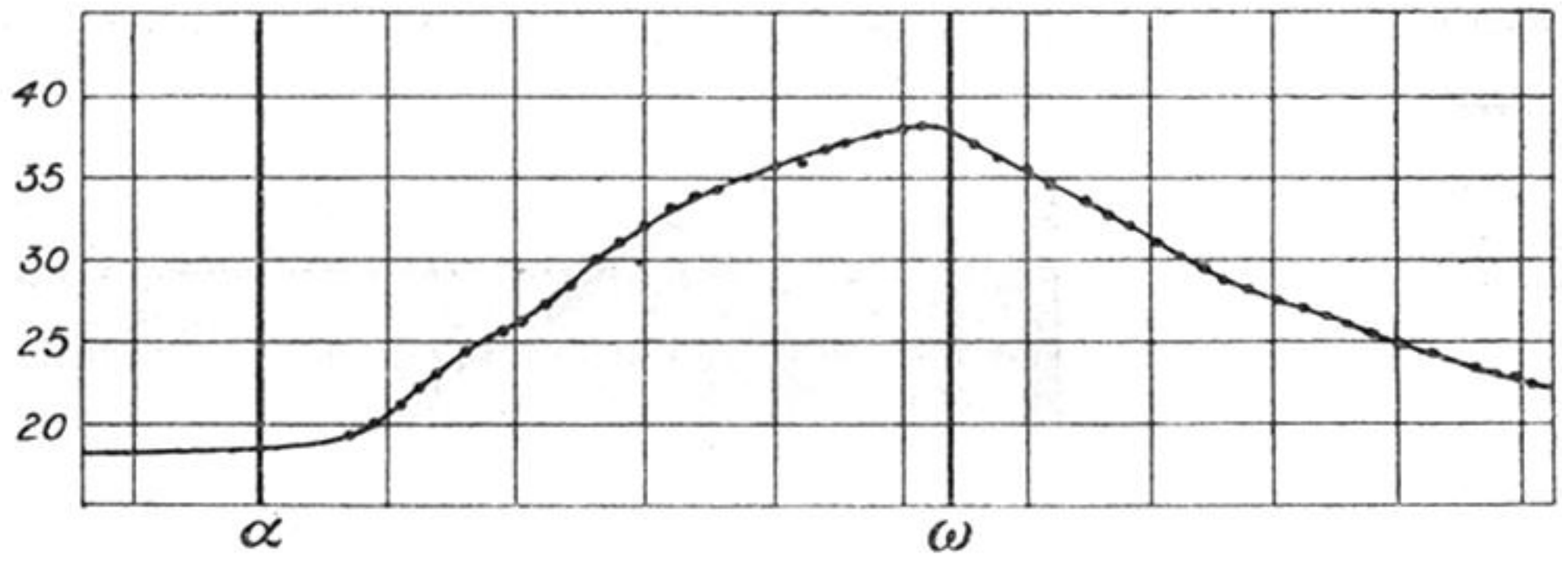
N.B.—In figs. 10 and 11 the current of injury was downwards or south, and falls upwards or north; the negative variation was upwards.

Fig. 11. Simultaneous record of the negative variation of nerve and of the contraction of muscle with increasing strength of stimulation (3 to 6 units of Berne coil).

The effect in nerve increases arithmetically with the stimulation, and continues to increase with increasing strength of stimulation beyond the point at which maximum tetanus of muscle is produced. The maximum contraction grows less in the progress of experiment.

Fig. 12. (Obs. 773.)



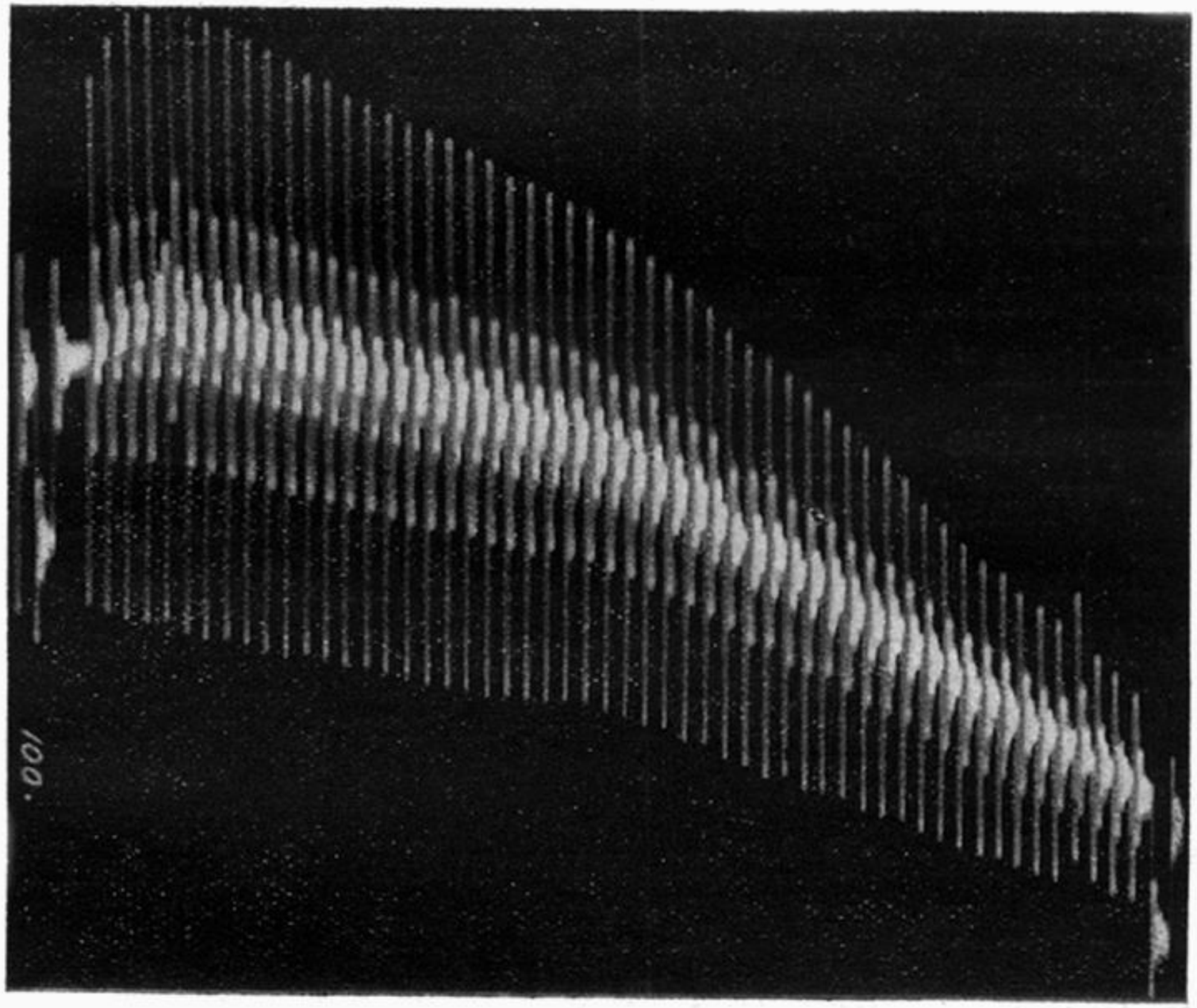


Effect of a rise of temperature from 18° to 38° upon the nerve response. The delay between the maximum temperature reading and the maximum diminution of the nerve response is probably owing to the "lag" of the apparatus between the nerve and the source of heat. The nerve was in a glass chamber in a copper oven, under which a small spirit-lamp flame was placed at α and removed at ω. The temperature chart below is that of corresponding readings each minute of a thermometer in the oven. No marked effect occurs with a rise of at least 10°; with a rise to 38°—viz., of 20°—the response diminishes; with the subsequent fall of temperature the response increases again.

The record is given in illustration of the fact that the response of fresh nerve is not affected by slight variations of temperature. The effects of considerable variations, as regards the character and sign of the response, will be described and considered in connection with the detailed study of positive and negative effects of excitation.

N.B.—The actual magnitudes of excursions, *e.g.*, in fig. 13, as compared with fig. 12, are of no account as indicative of magnitudes of nerve-effects in terms of potential. The resistance differs in different nerves, and the galvanometer is more or less shunted. The magnitudes of excursions must in all cases be compared with the magnitude of the standard excursion by 0·001 volt taken at beginning and end of each observation.

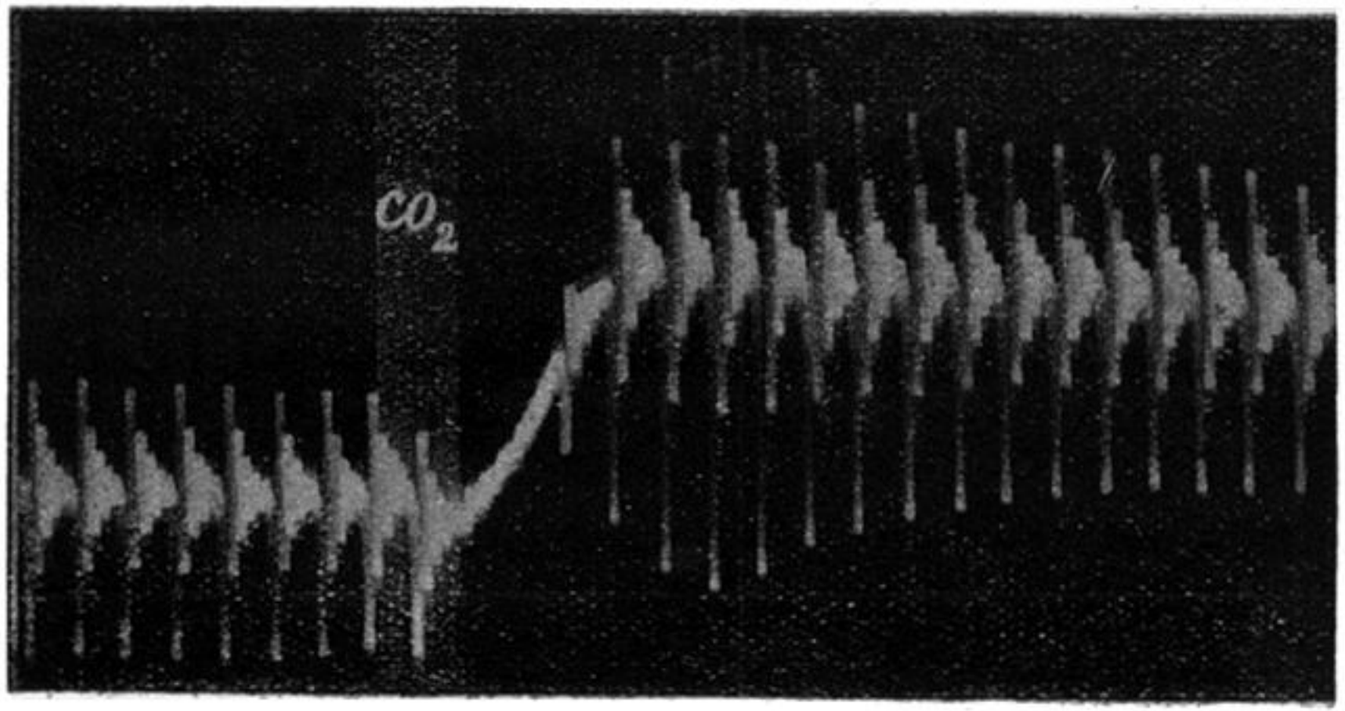
Fig. 13. (Obs. 825.)



Effect of drying by evaporation upon the response of nerve. Stimulation as usual, 10 units tetanisation for $\frac{1}{8}$ minute at each minute.

Nerve kept in physiological saline for $2\frac{1}{2}$ hours after excision; lid of gas-chamber removed. In the absence of evaporation such a nerve would probably have given an uniform series, as in fig. 6. The relative magnitudes of the deflections by 0·001 volt at beginning and end of the series indicate to what extent the diminishing deflection is due to increasing resistance of the nerve.

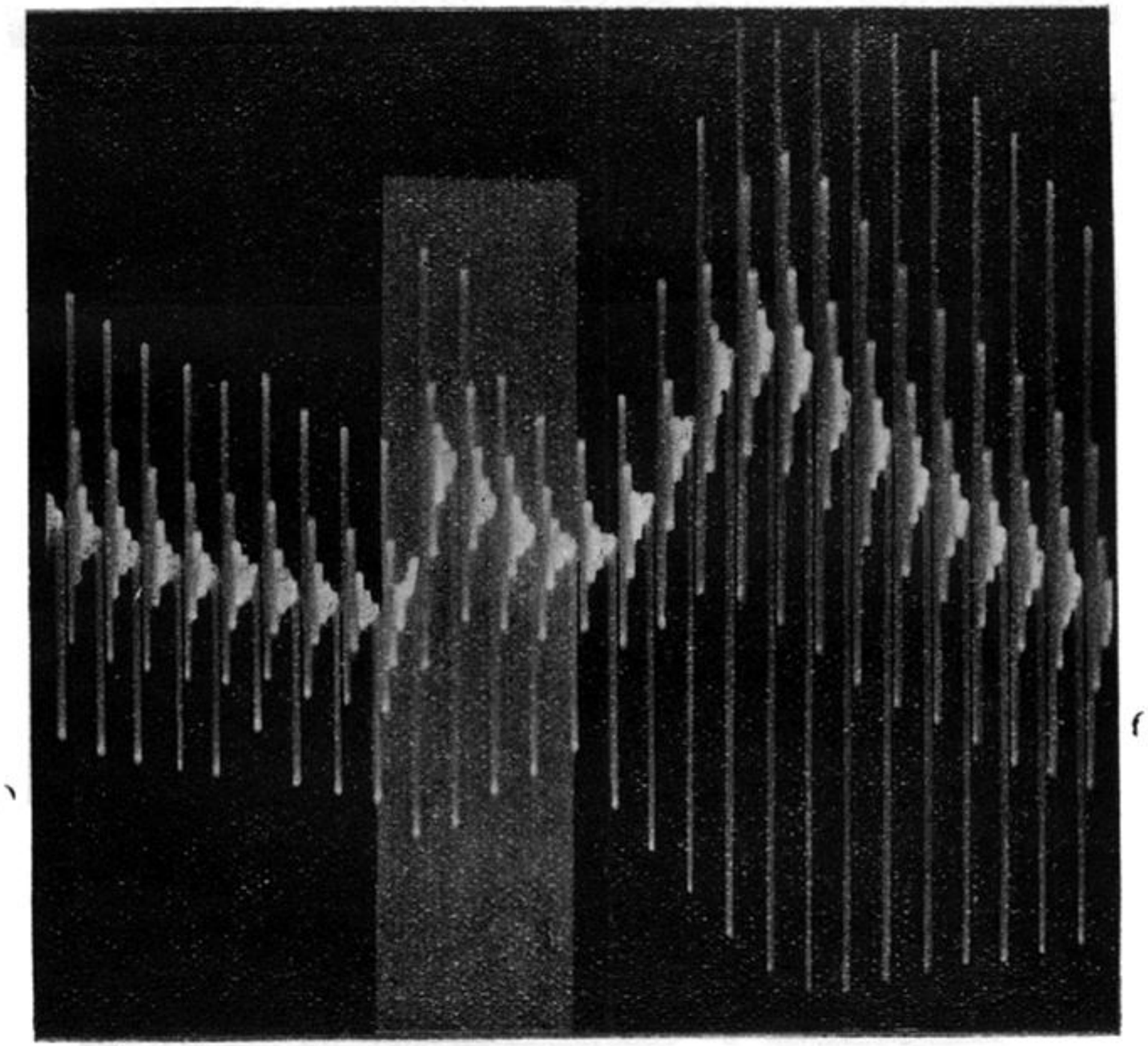
Fig. 14. (Obs. 200.)



Effect of CO_2 in full stream on normal freshly excised nerve. "Typical effect," viz., primary abolition followed by secondary augmentation of excitability.

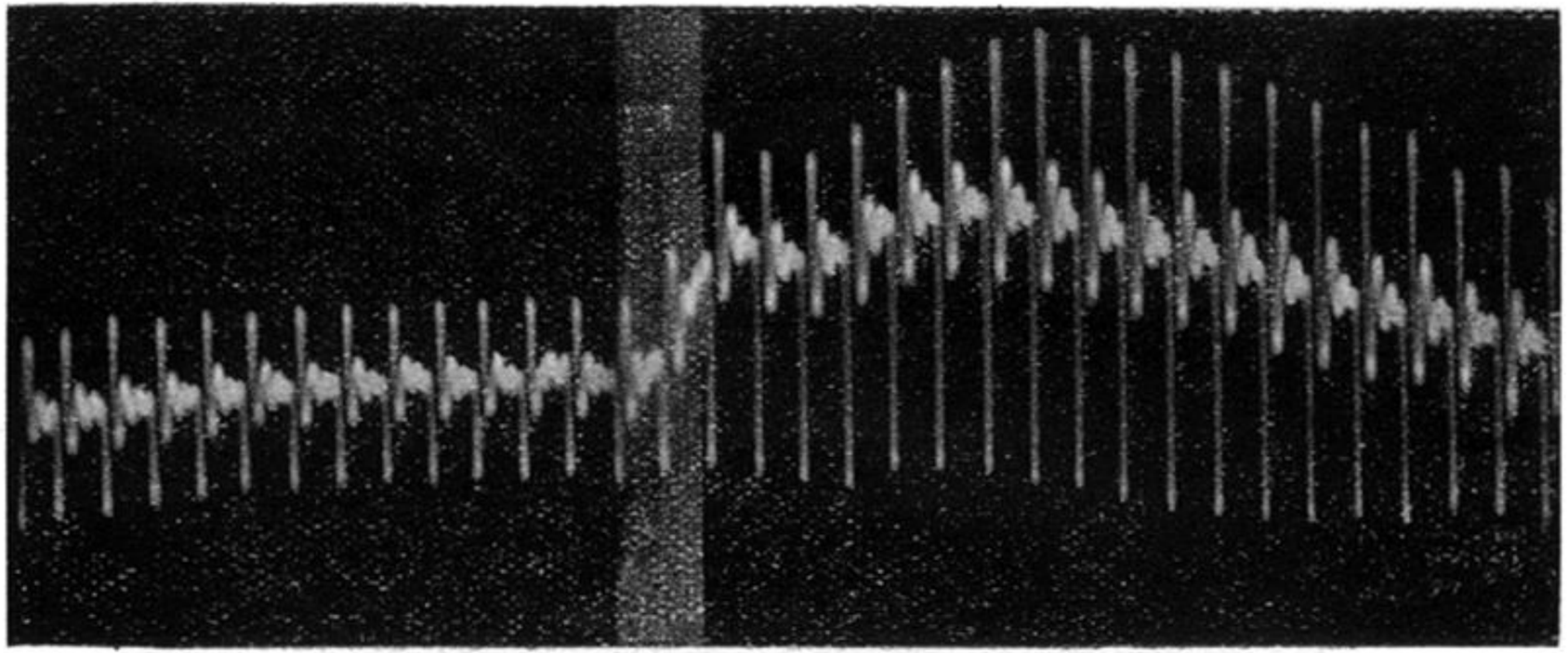
The gas-chamber containing the nerve measures 30 cub. centims. The nerve is tetanised each minute for $7\frac{1}{2}$ seconds by ordinary make and break shocks at a frequency of about 70 per second, and a strength of 10 units of a Berne coil, supplied by two Leclanché cells. Interrupter in primary circuit. Time of passage of CO_2 through the nerve chamber indicated by the vertical bar across the record. Deflection before CO_2 = that of 0·0006 volt; after $CO_2 = 0$; 6 minutes later = 0·0012 volt. The "lag" of the CO_2 effect is probably due to diffusion time of the gas in contact with the nerve.

Fig. 15. (Obs. 537.)



Effect of a "moderate" quantity of CO_2 on nerve under similar conditions. Primary augmentation, diminishing; secondary further augmentation.

Fig. 16. (Obs. 869.)



Effect of a "small" quantity of CO_2 on nerve under similar conditions. Primary augmentation, in this instance, checked by a very slight diminution. Compare with figs. 5 and 6.

Fig, 17. (Obs. 633.)

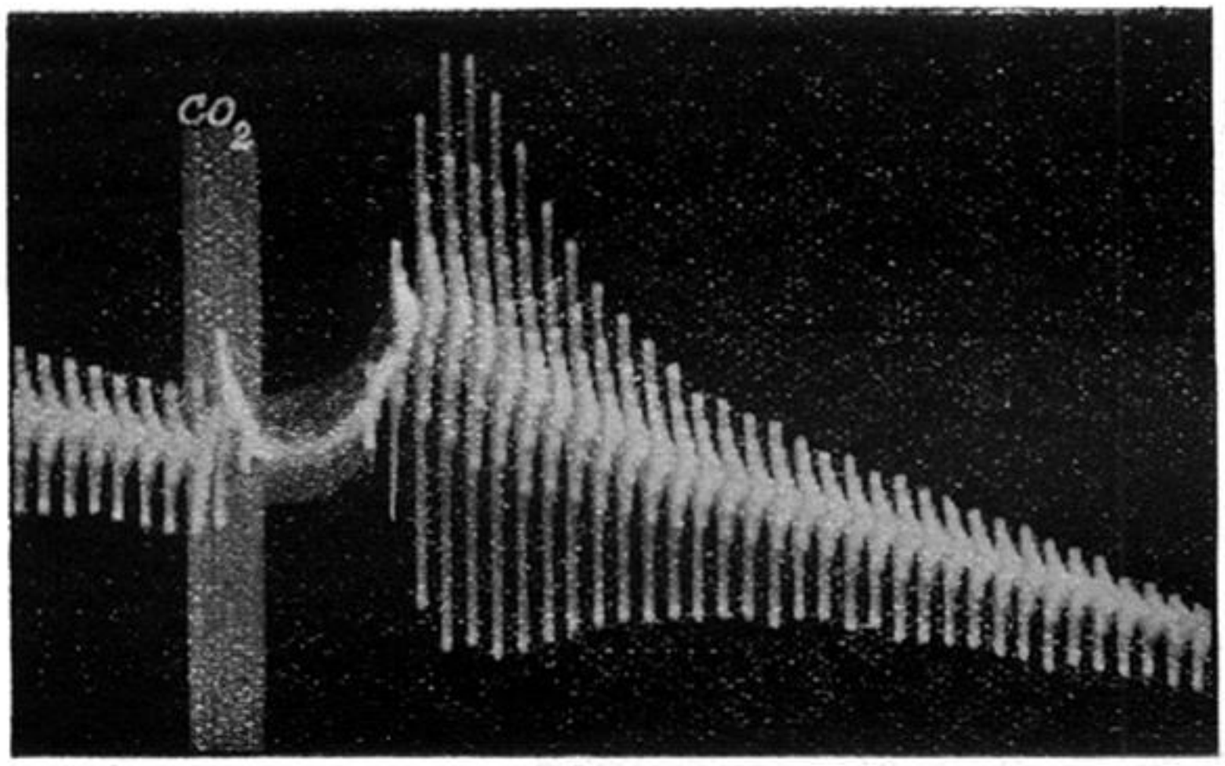


Fig. 18. (Obs. 652.)

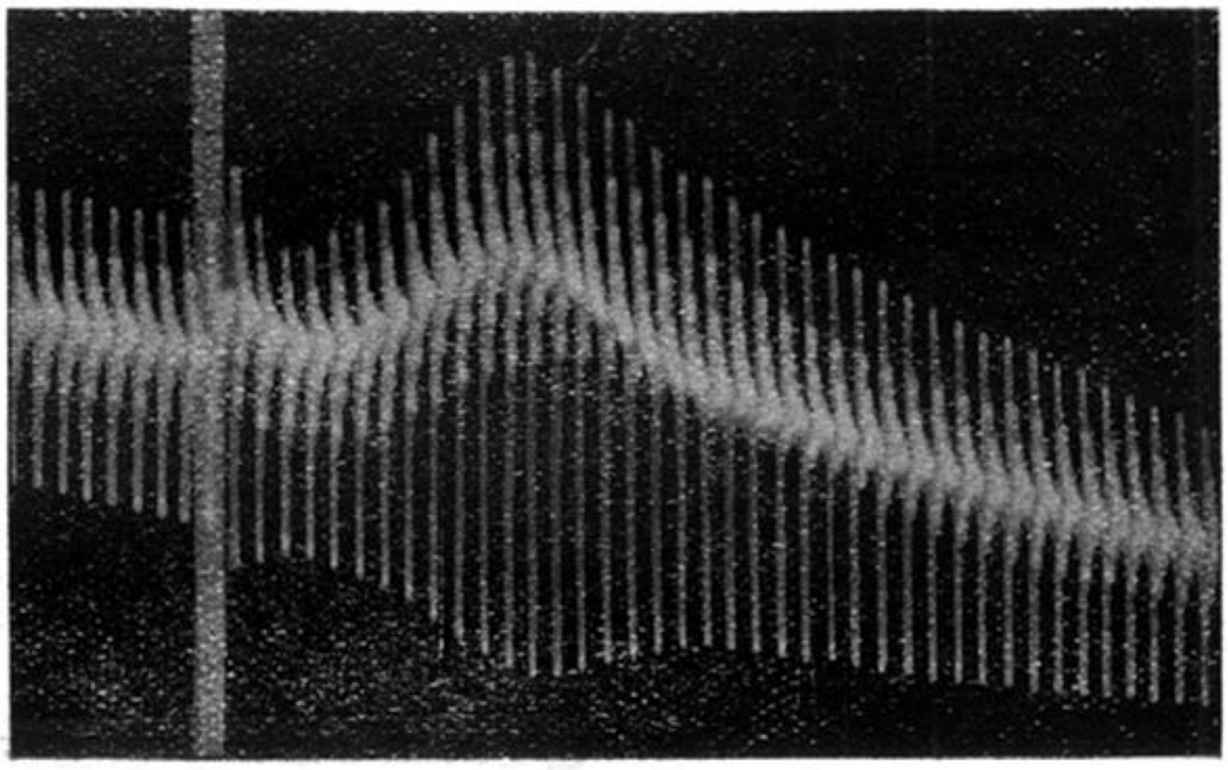
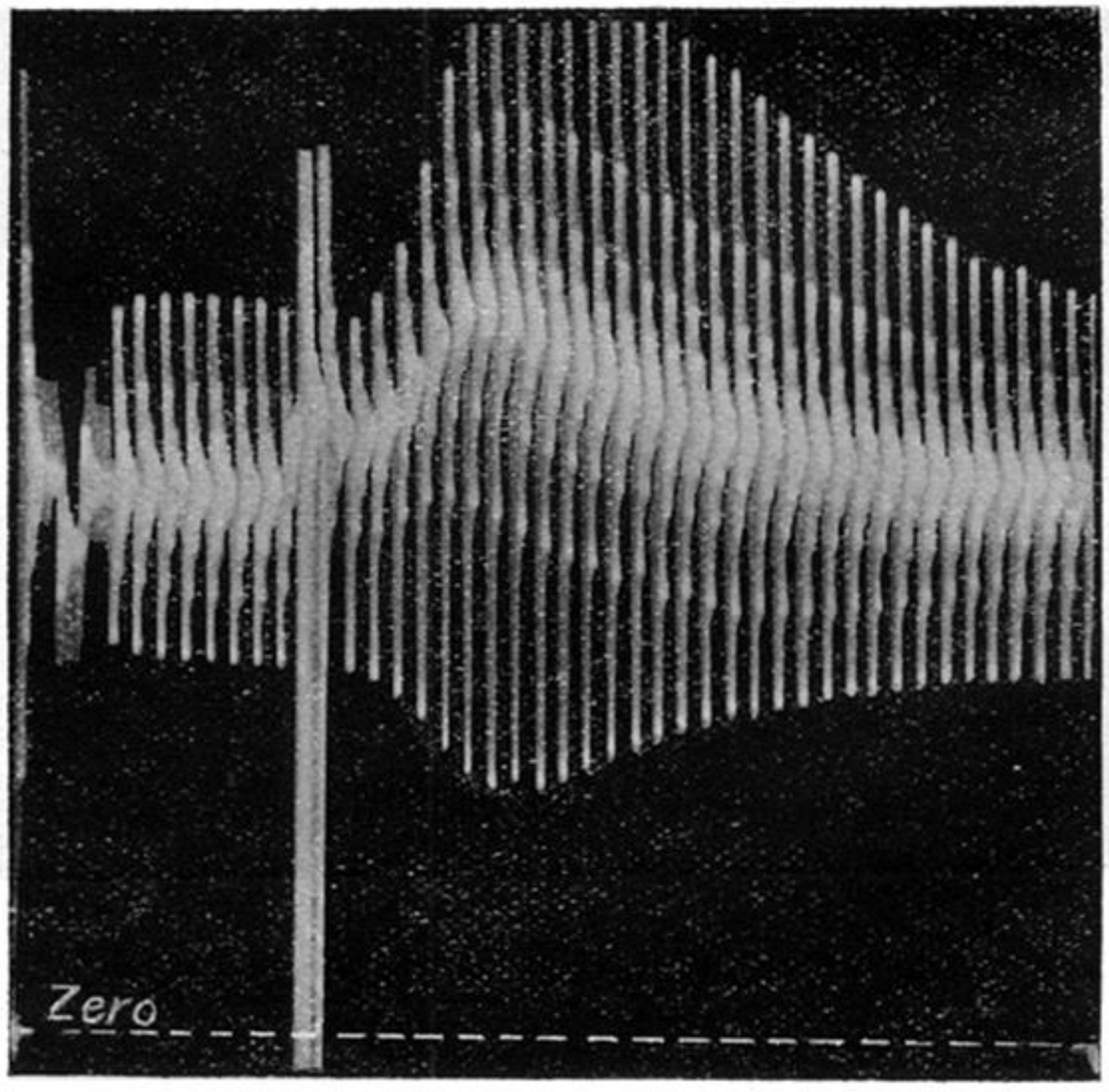
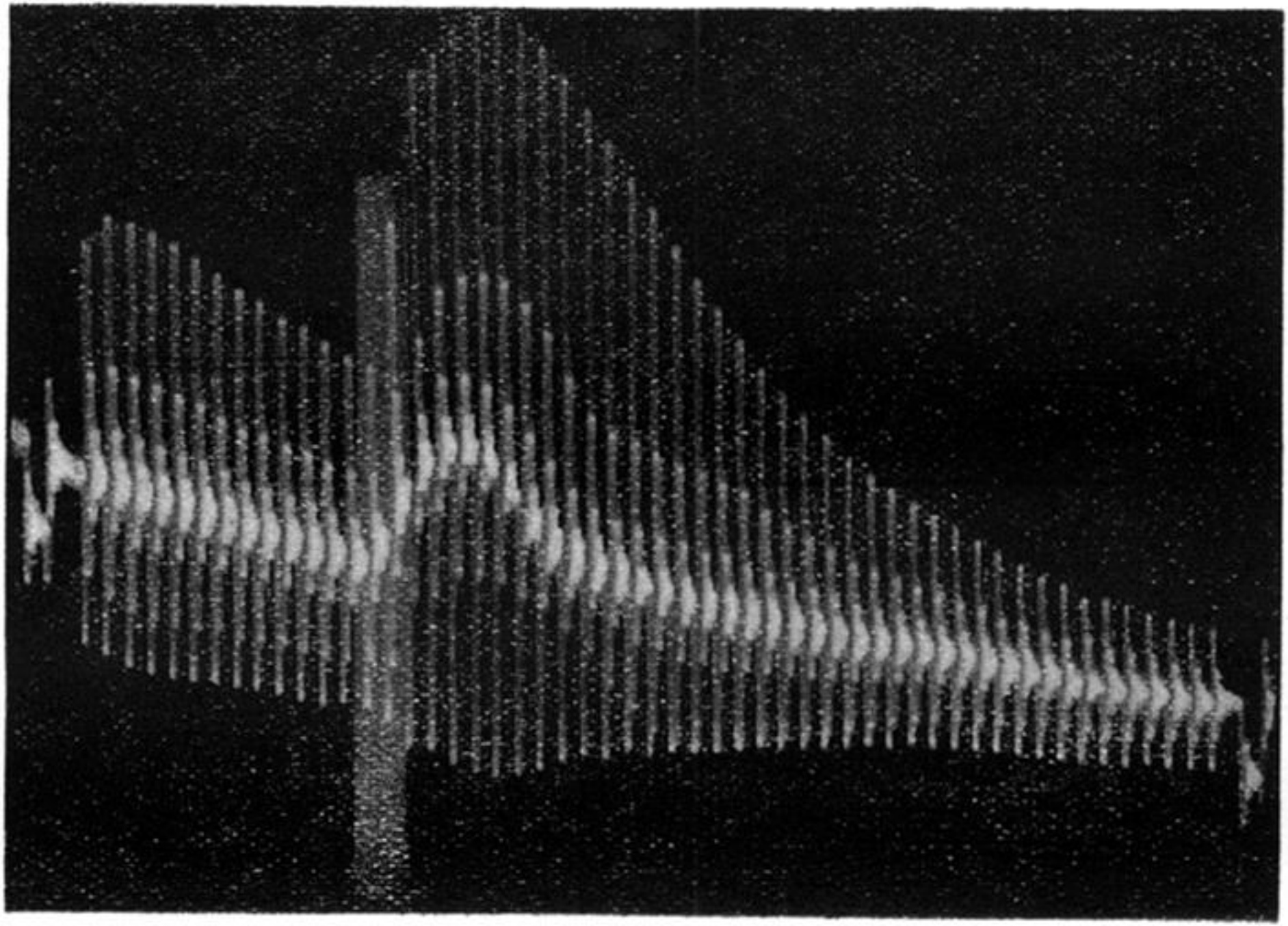


Fig. 19. (Ob. 674.)



Figs. 17, 18, and 19. "Full," "medium," and "slight" effects of CO_2.

Fig. 20. (Obs. 589.)



Effect of expired air.

Fig. 21. (Obs. 671.)

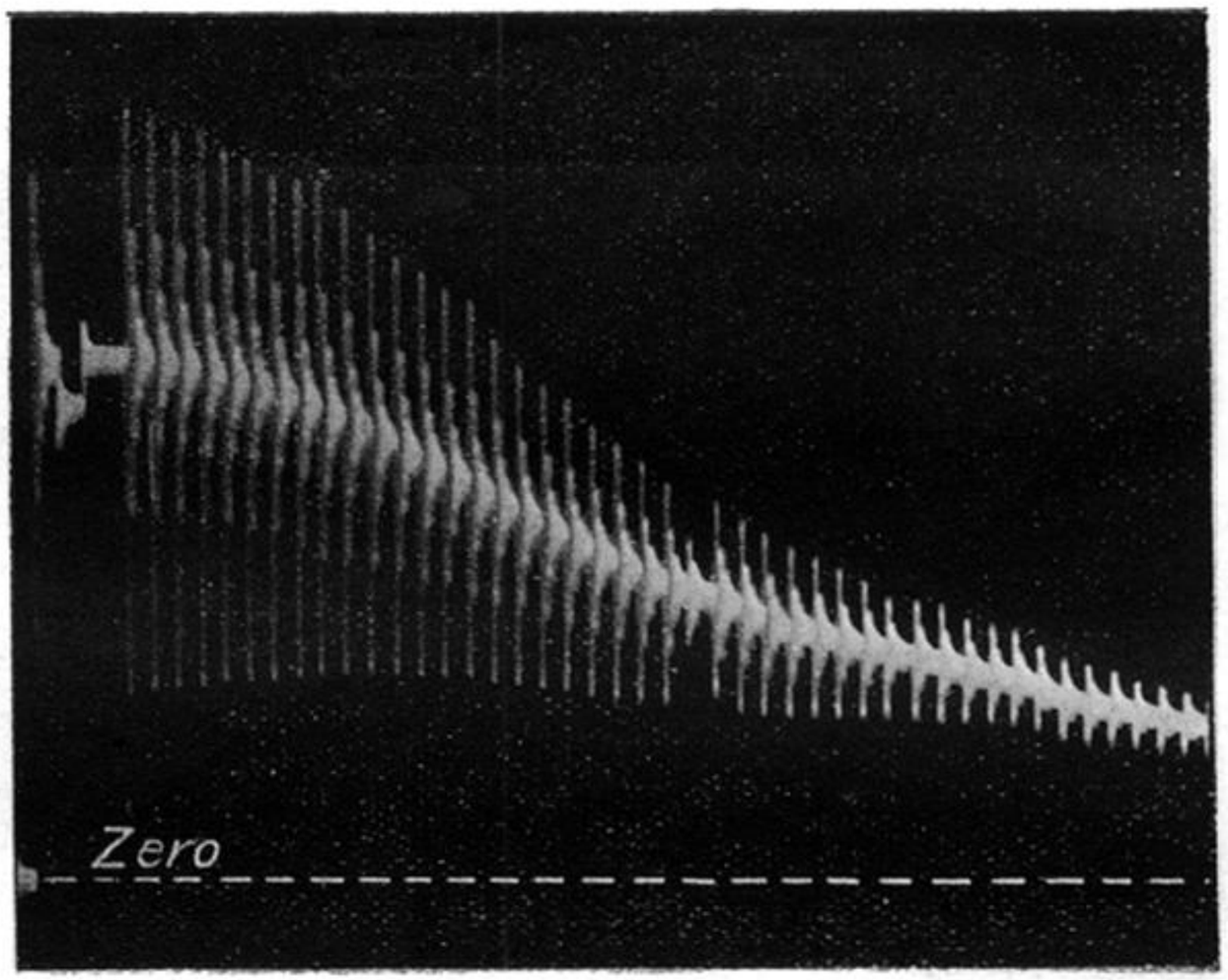
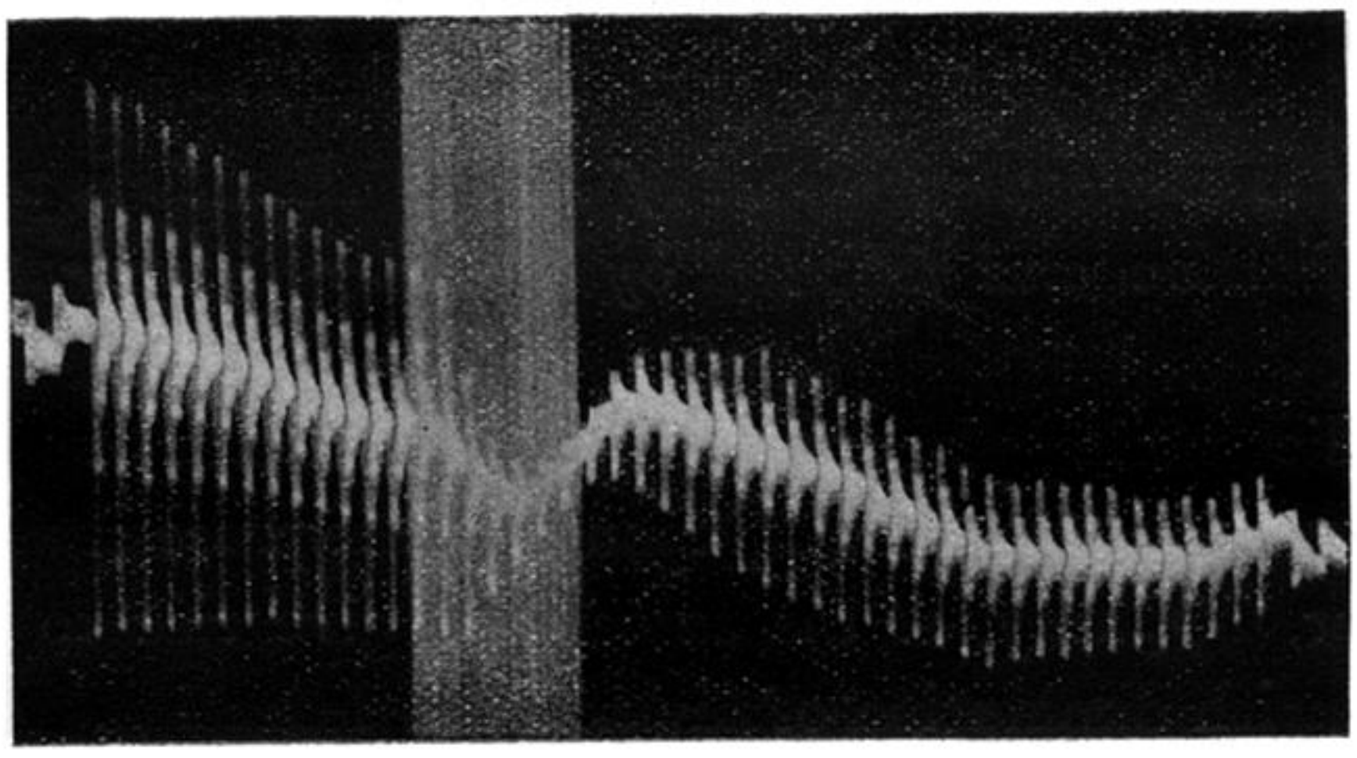
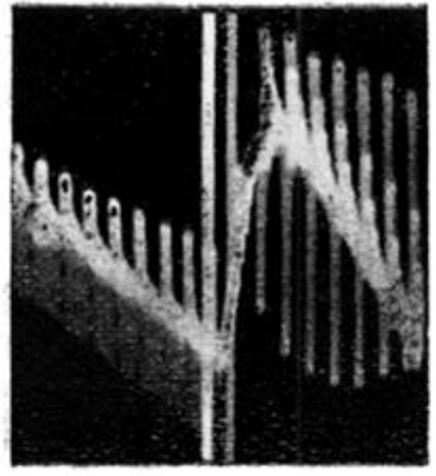


Fig. 22. (Obs 672.)



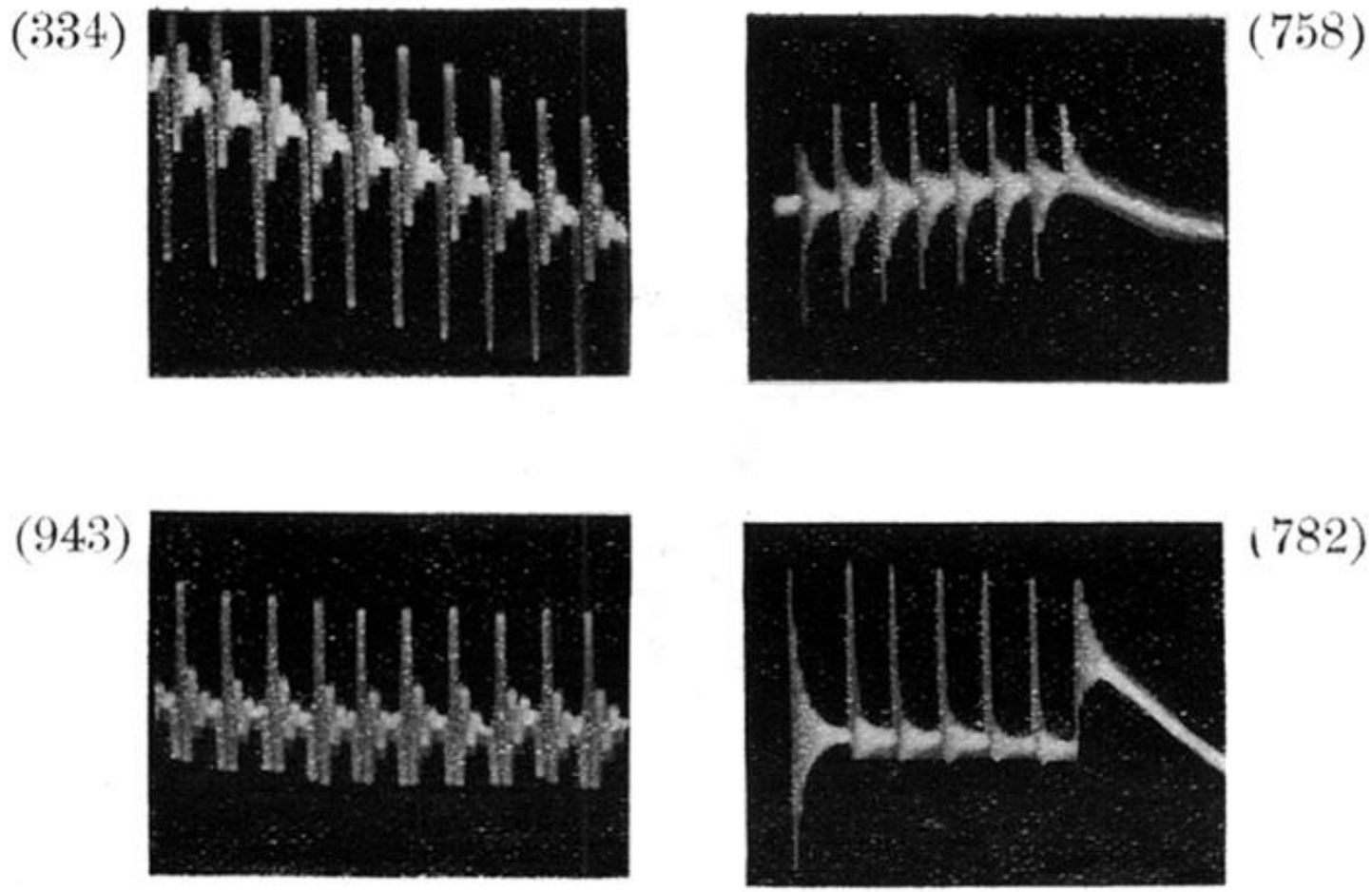
Records characteristic of nerve "carbonised" by prolonged sojourn in the living tissues of a pithed animal. The deflection is at first much larger than usual and declines more rapidly (fig. 21), the abolition caused by CO_2 is more prolonged than usual, and the after-augmentation is comparatively insignificant (fig. 22).

Fig. 23. (Obs. 788.)



Currents of action, and effect upon them of CO_2, with the exciting and galvanometer circuit conjoined (as described on pp. 12, 13).

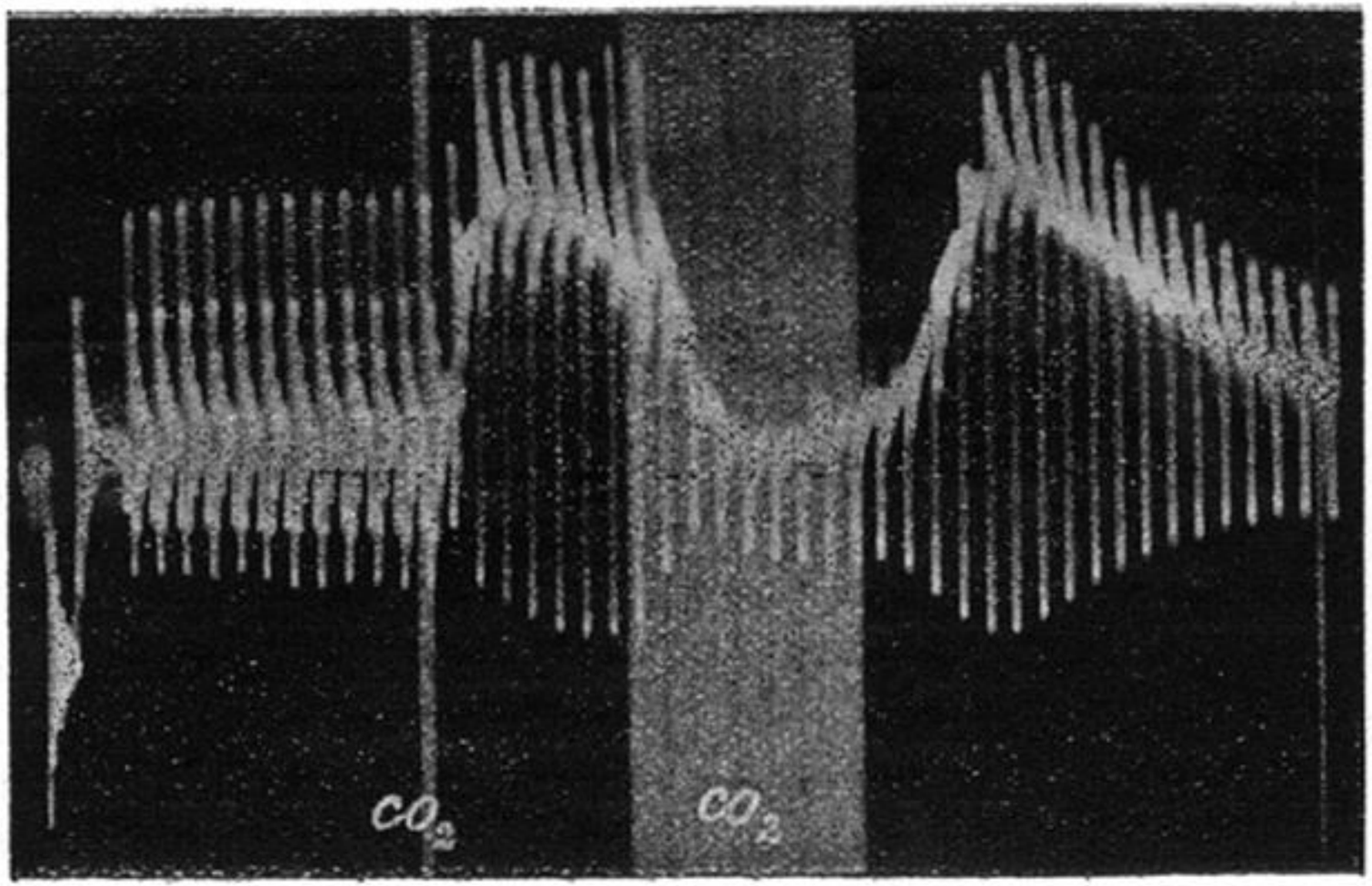
Fig. 24.



Observations with the partially damped galvanometer, in 334 and 943, with an oscillation period of $12\frac{1}{2}$ secs., and a swing-back $= \frac{2}{3}$ the original swing; in 758 and 782, with an oscillation period of $8\frac{1}{2}$ secs., and a swing-back $= \frac{1}{3}$ the original swing.

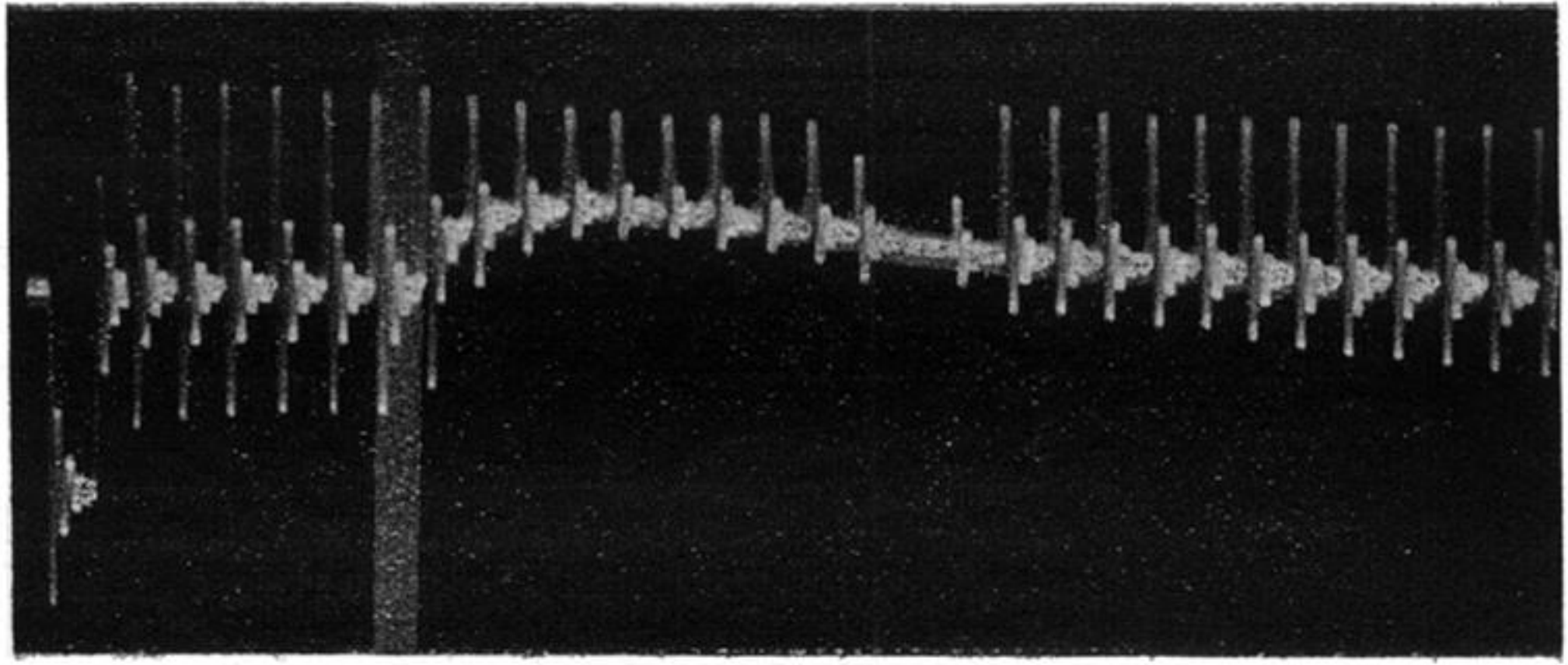
No.	Effect.	Uncorrected after-effect.	Correction.	Corrected after-effect.
334	−10	+6·6	+6·6	0
943	− 4·5	+7	+3	+4
758	+ 6	−6	−2	−4
782	+12	−1	−4	+3

Fig. 25. (Obs. 734.)



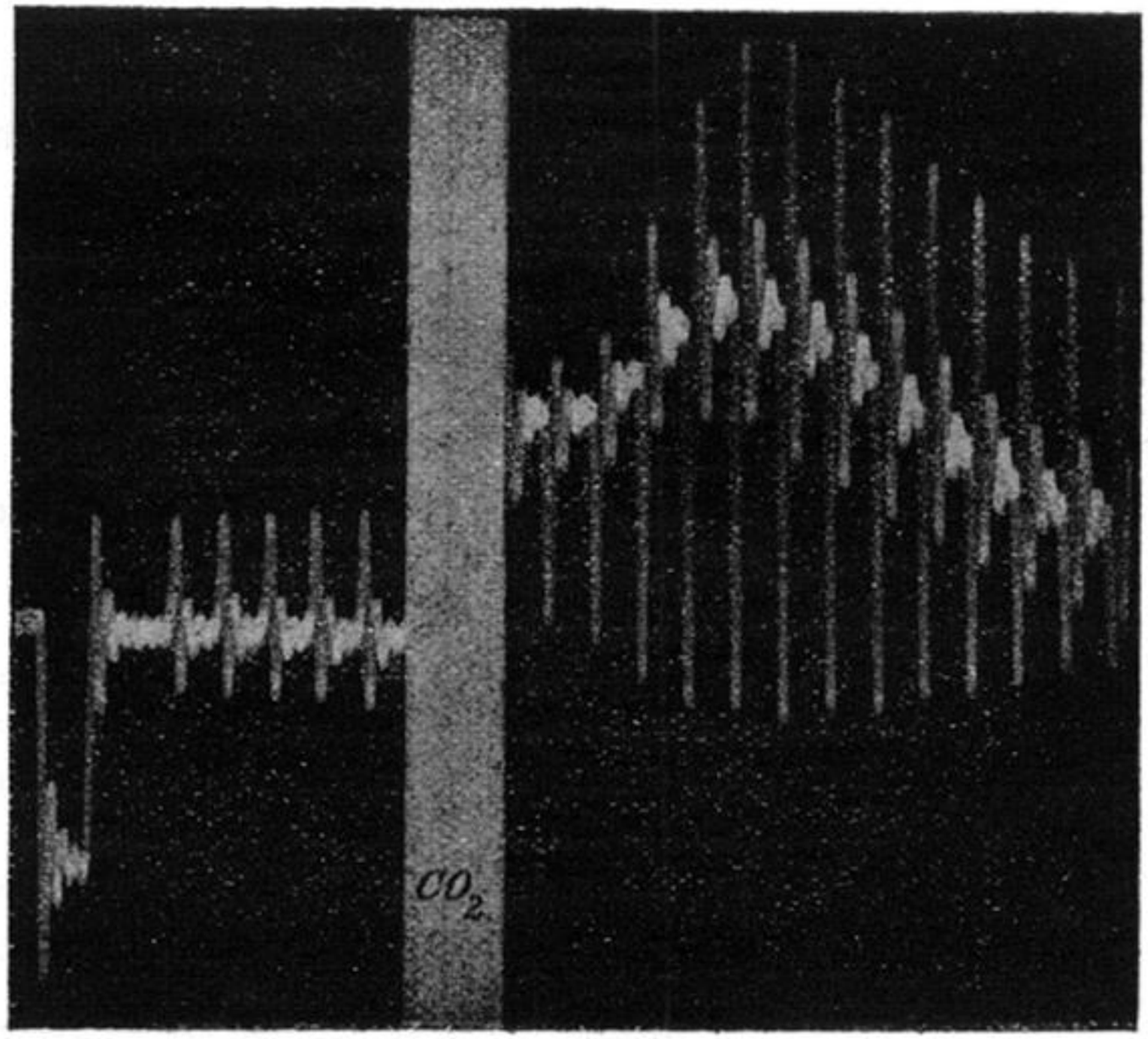
Influence of "little" and "much" CO_2 upon nerve in the second stage. (See Table B.)

Fig. 26. (Obs. 982.)



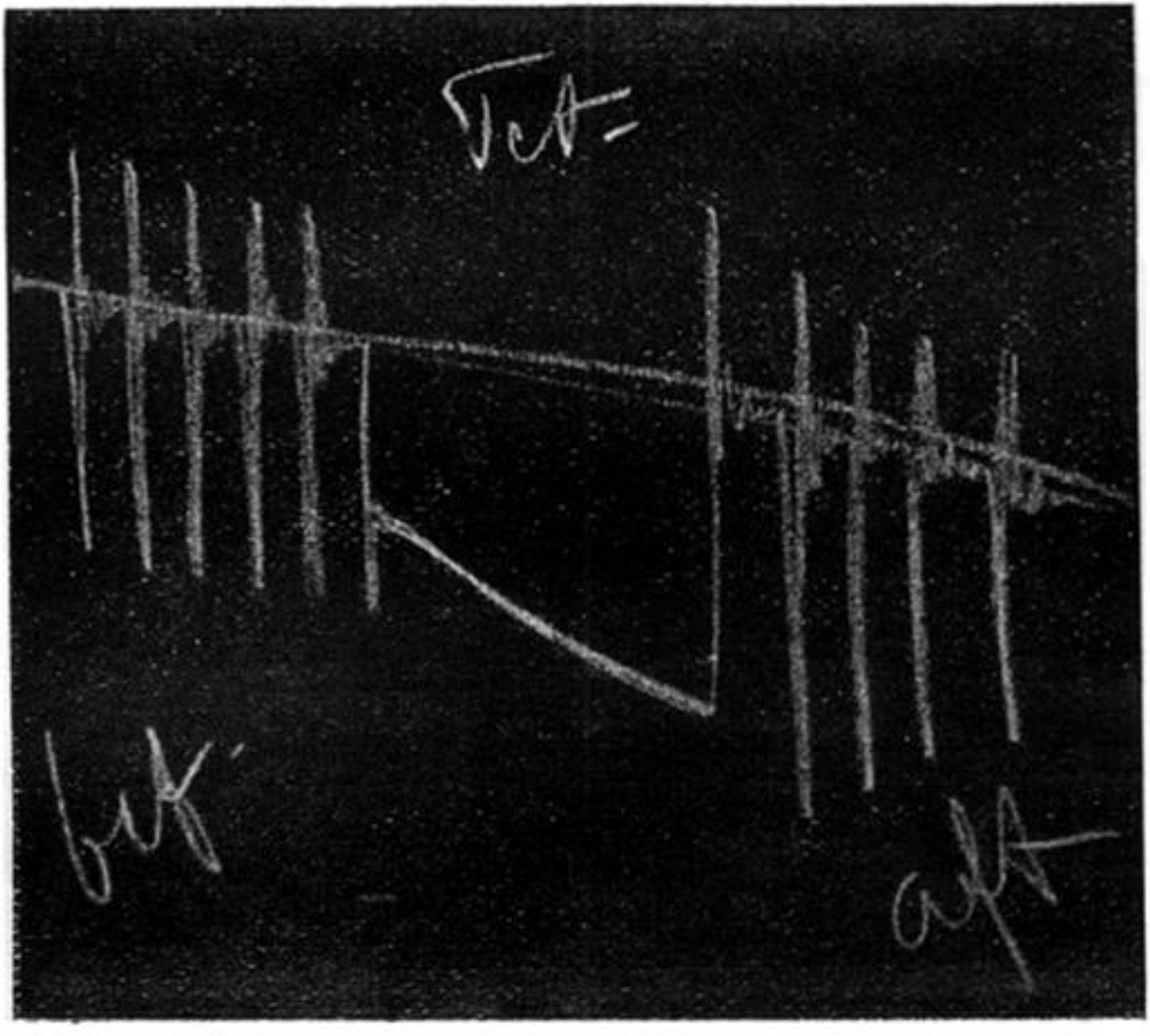
Influence of CO_2 upon nerve in the third stage; diminution of the positive effect.

Fig. 27. (Obs. 881.)



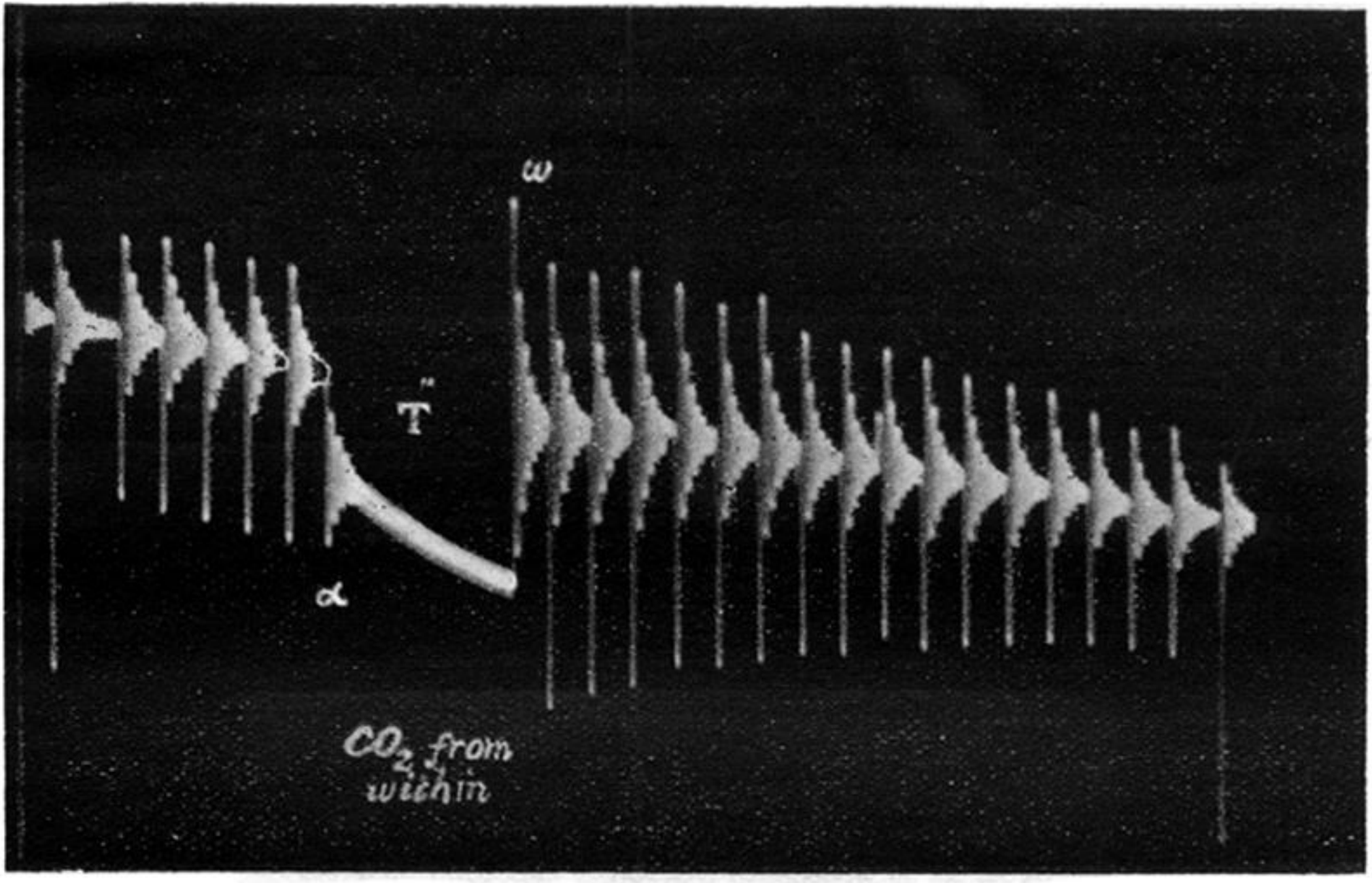
Influence of CO_2 upon nerve in the third stage; reversal of effect from positive to negative.

Fig. 28.



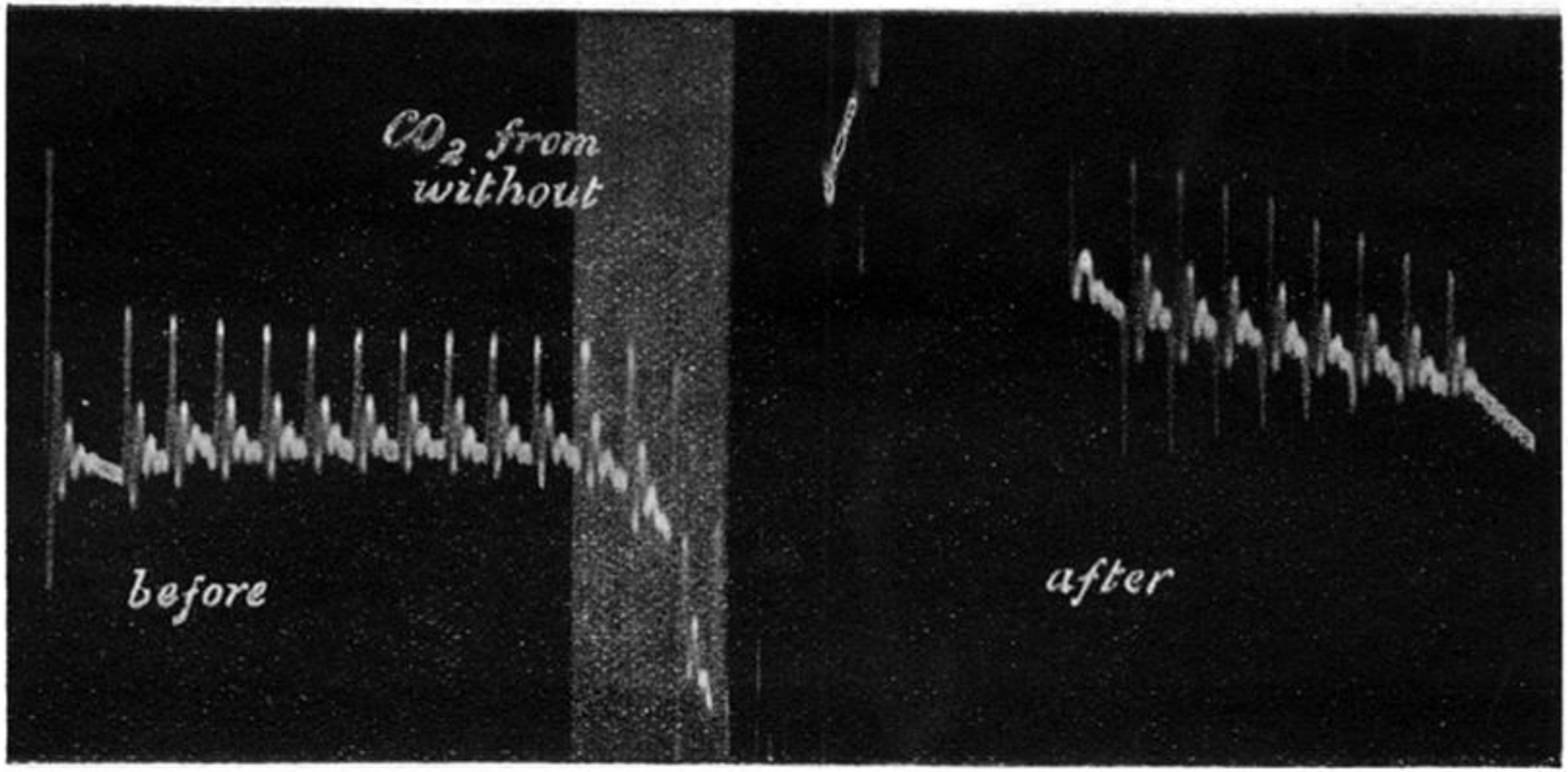
Hypothesis.

Fig. 29. (Obs. 675.)



Verification.

Fig. 30. (Obs. 859.)



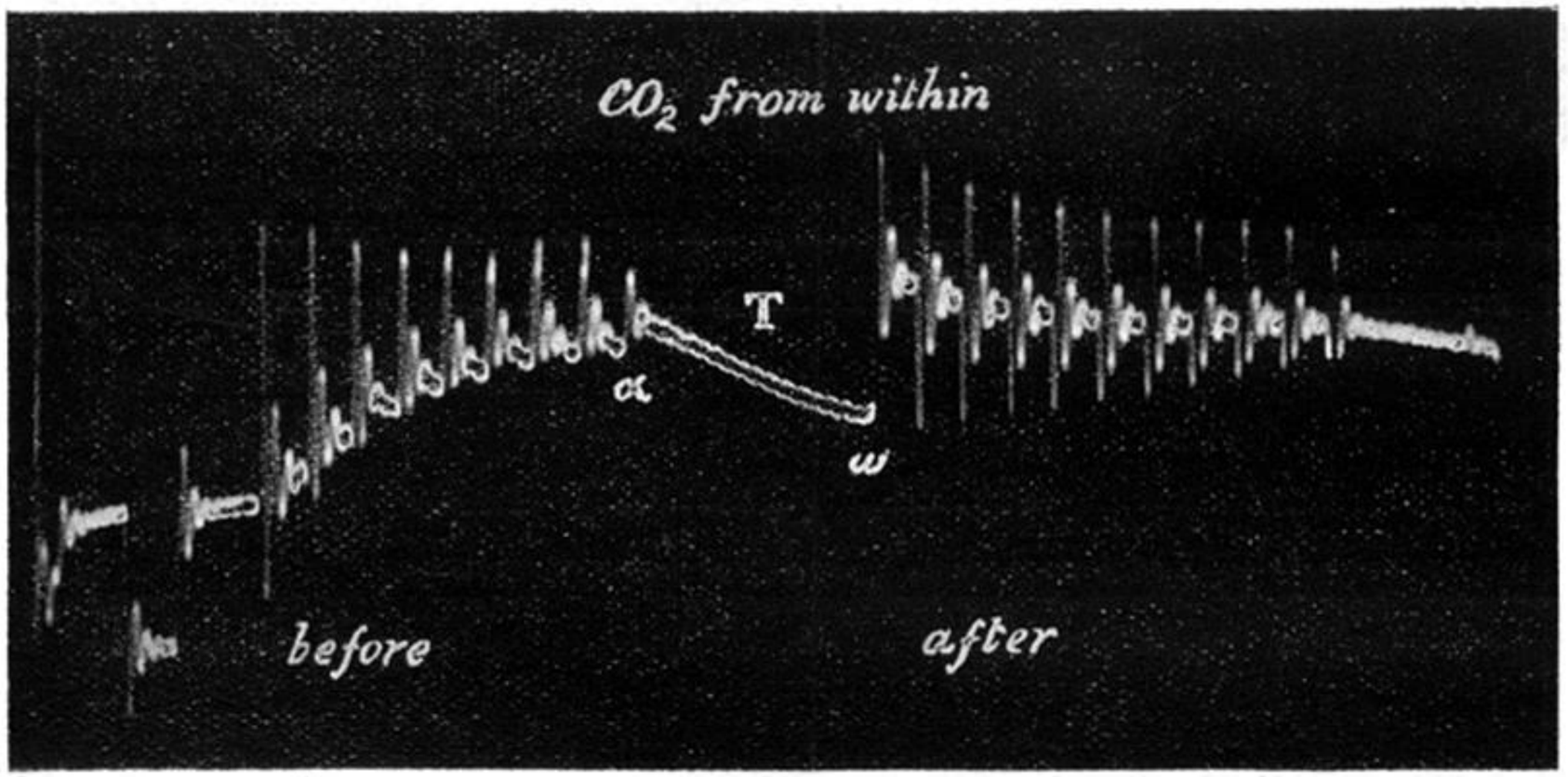
Nerve in third stage after half an hour's rest. Influence upon the deflection of a slow stream of CO_2.

Before CO_2.—The deflections are north north, consisting of a positive effect followed by a positive after-effect.

During the passage of CO_2 the spot fell off plate south. Subsequently, having been readjusted north, it rose off plate north.

After CO_2.—Large negative effect diminishing, smaller positive after-effect increasing. Half an hour later the deflections were again north north.

Fig. 31. (Obs. 858.)



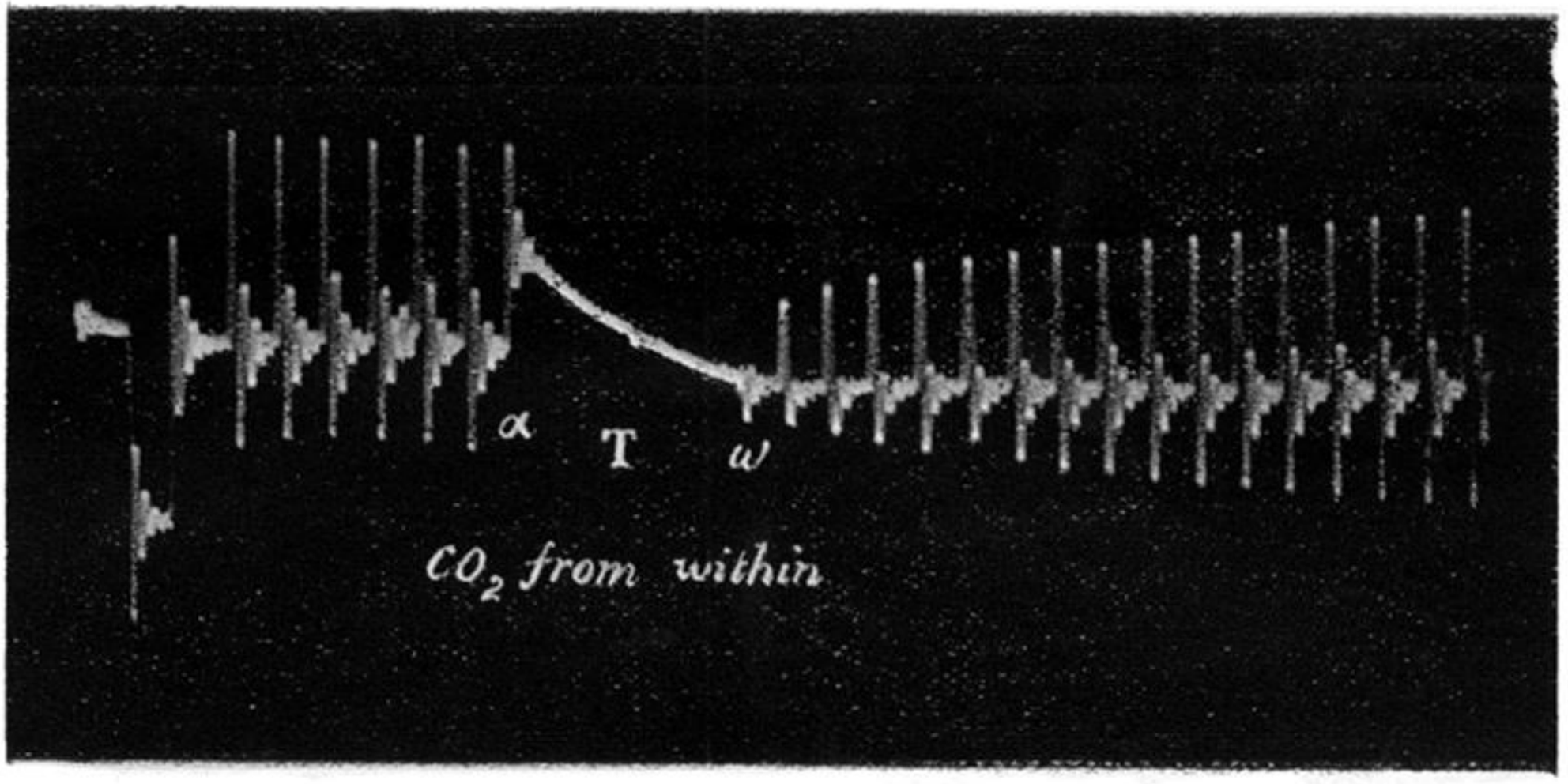
Same nerve giving a positive effect, followed by a positive after-effect. North north.

Before Tetanus.—Positive effects rapidly diminishing. Positive after-effects summating and causing the base line to rise. The last three effects exhibit a slight negative effect at the end of each excitation, interposed between the positive effect and the positive after-effect.

During Tetanus.—Positive deflection becoming increasingly negative. The first deflection at the commencement of the tetanus is north, the first deflection at the end of the tetanus is north.

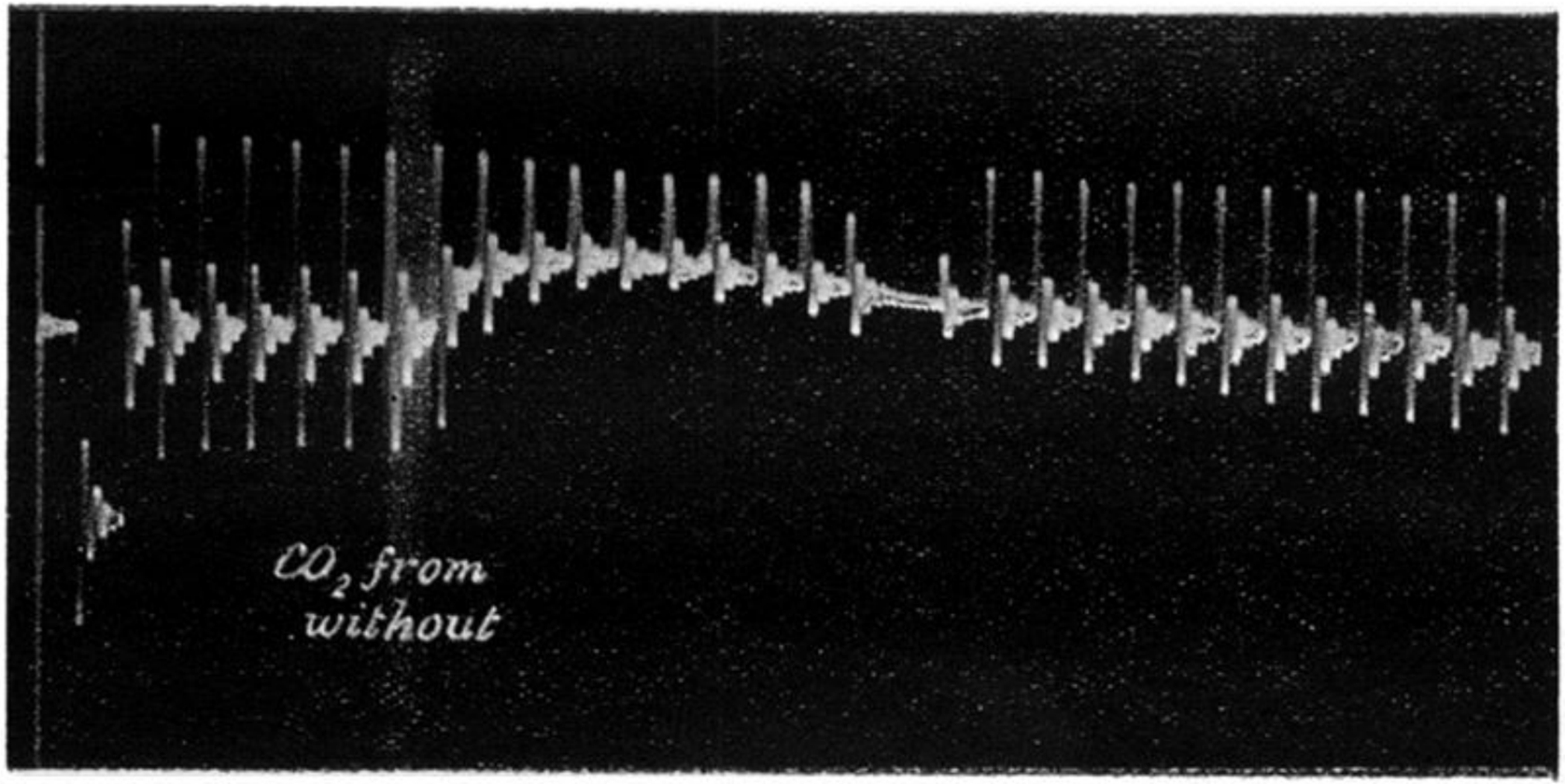
After Tetanus.—Large negative effects diminishing, smaller positive after-effects increasing. After a subsequent half hour of repose, the negative effects had vanished, the deflection had again become north north.

Fig. 32. (Obs. 984.)



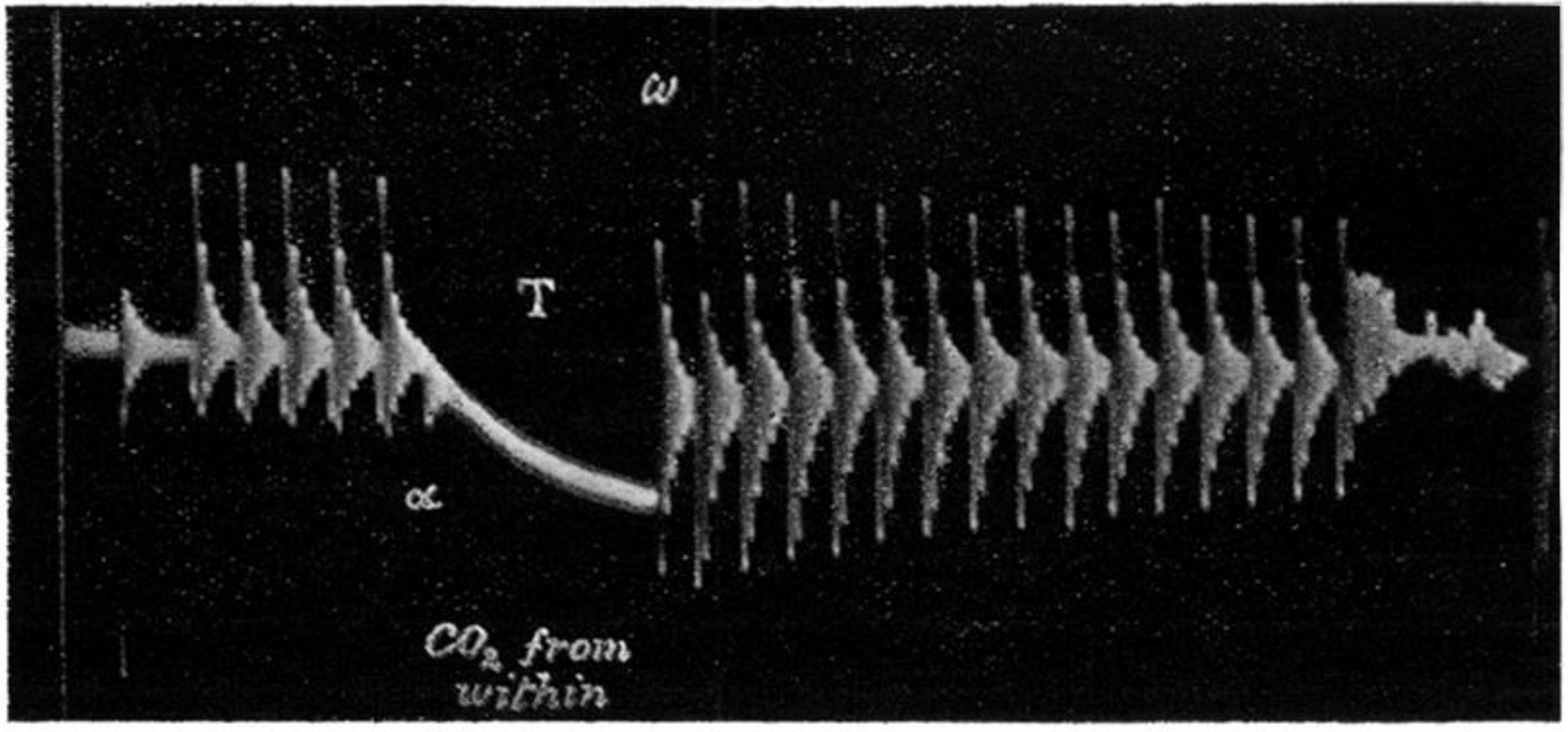
Influence of tetanisation upon nerve in the third stage; diminution of the positive effect.

Fig. 33. (Obs. 982.)



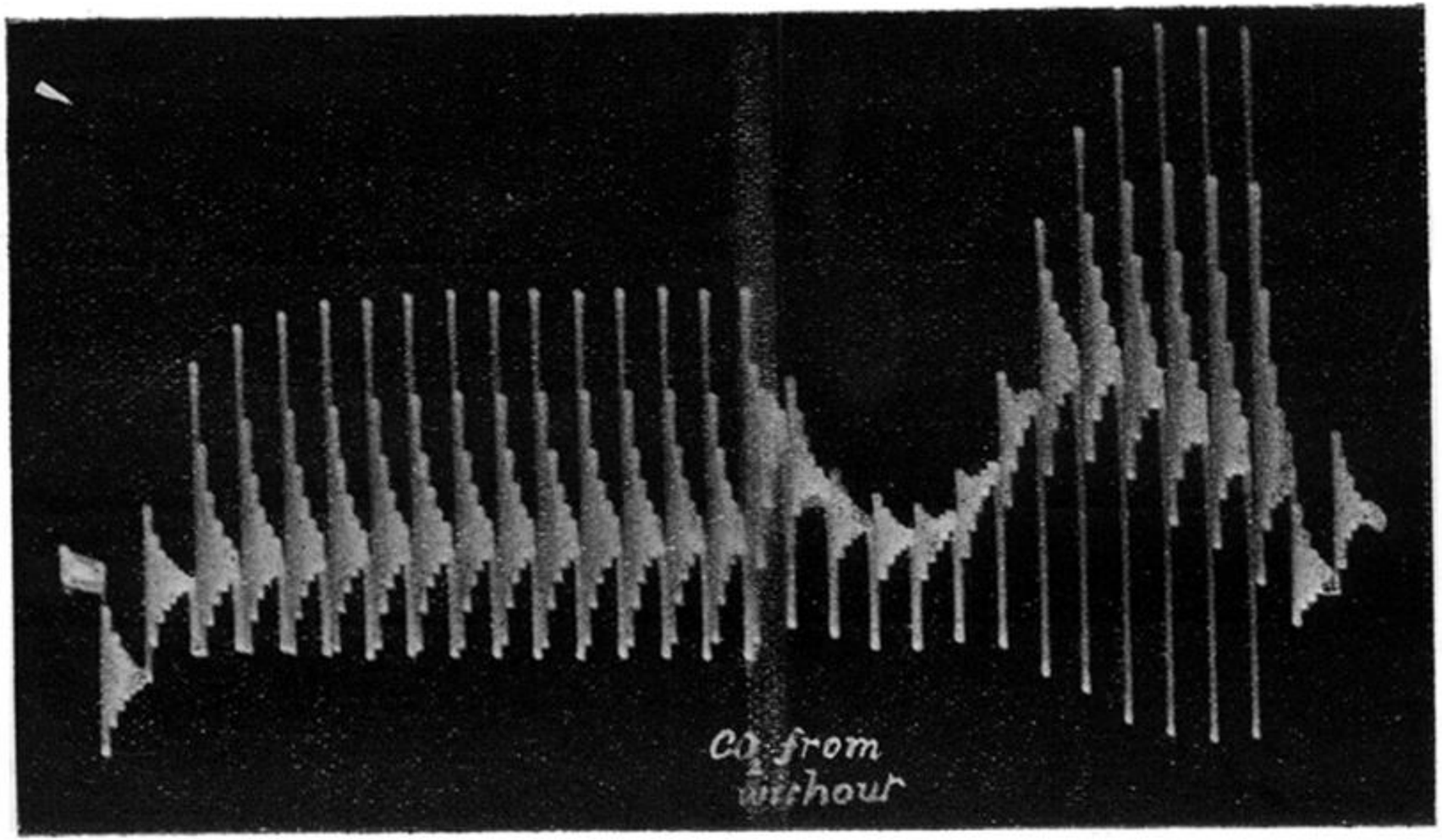
Influence of carbon dioxide upon nerve in the third stage; diminution of the positive effect. (The irregularity of response from the 16th to 18th minute is due to irregularity of the stimulating apparatus.)

Fig. 34. (Obs. 762.)



Influence of tetanisation upon nerve in the second stage, giving the response *s*.N., viz., small negative effect followed by large positive effect; augmentation of the negative effect.

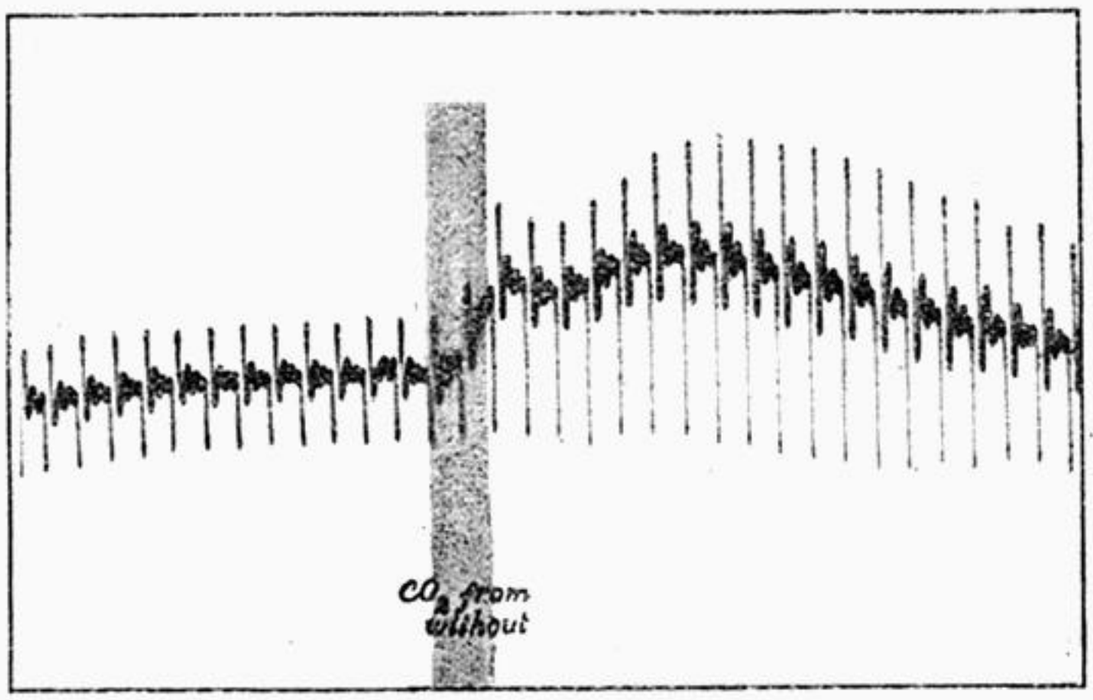
Fig. 35. (Obs. 710.)



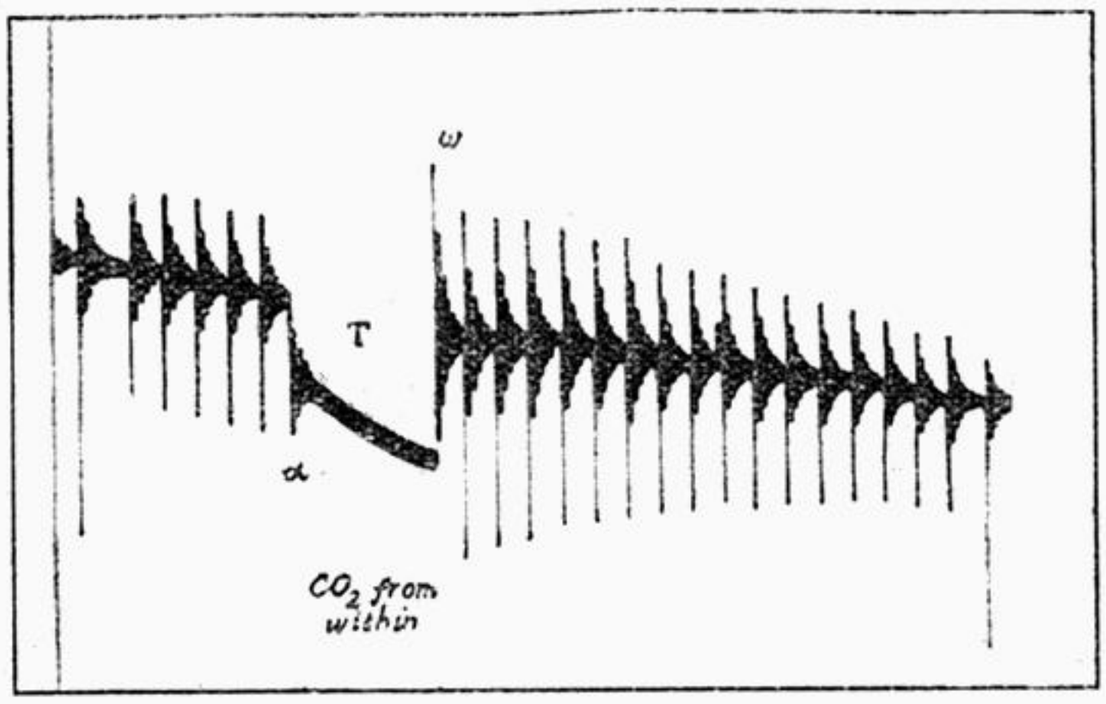
Influence of carbon dioxide upon nerve in the second stage, giving the response *s*.N.; augmentation of the negative effect.

Fig. 35A.

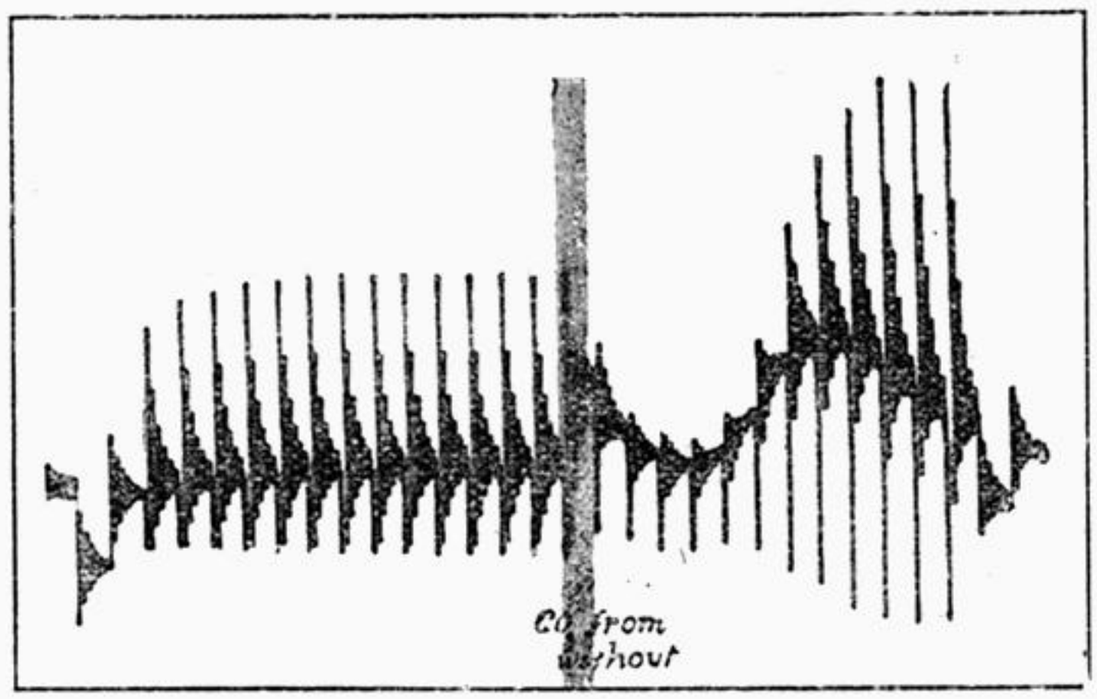
869



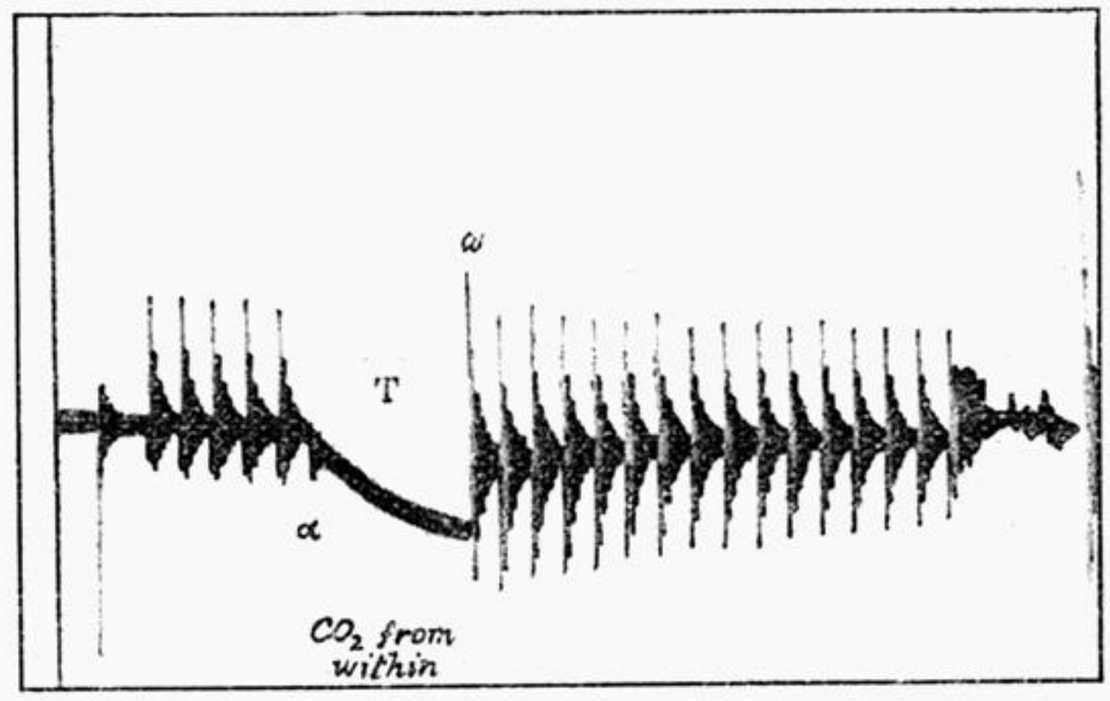
675



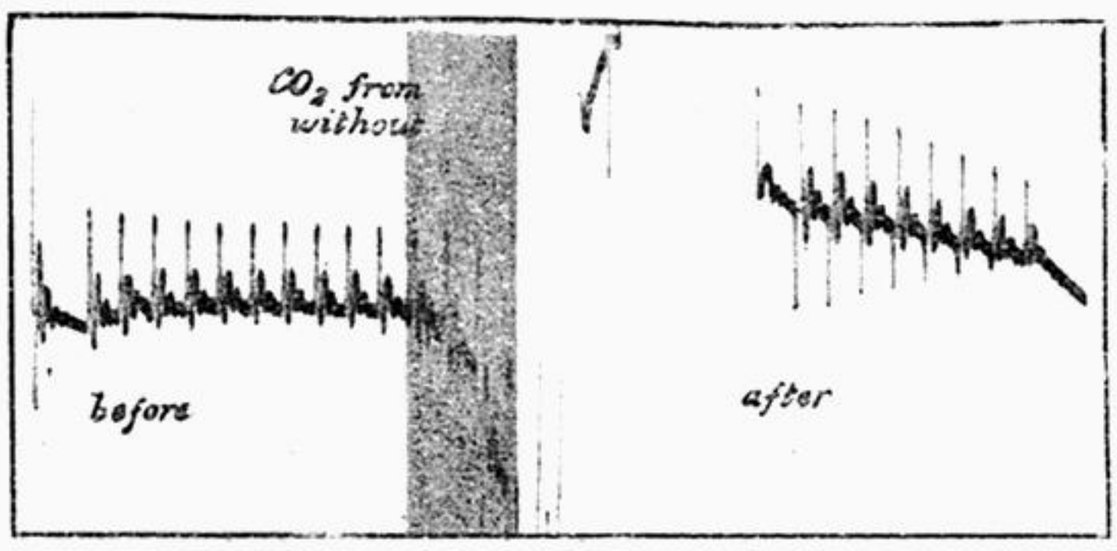
710



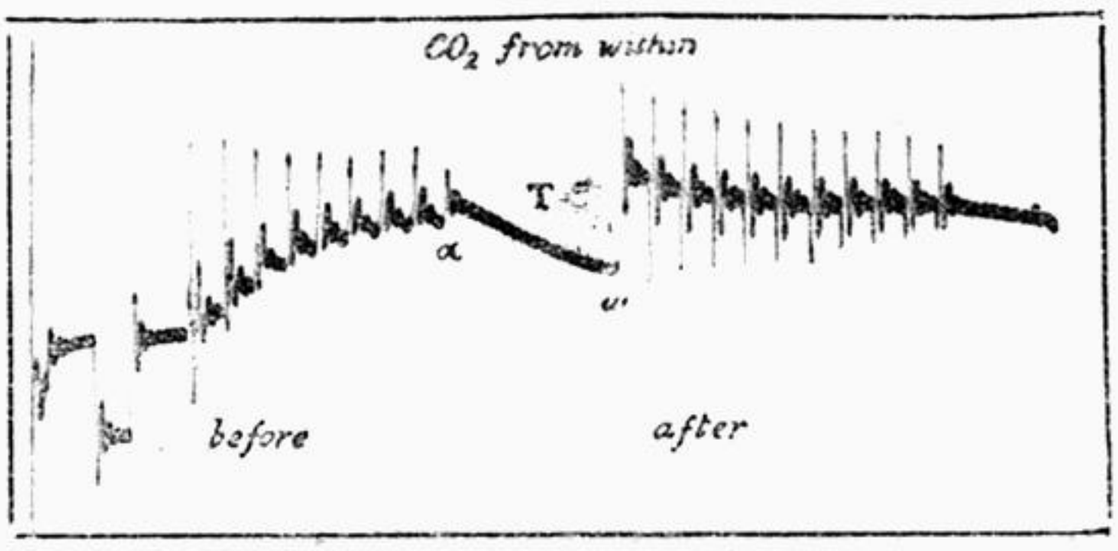
762



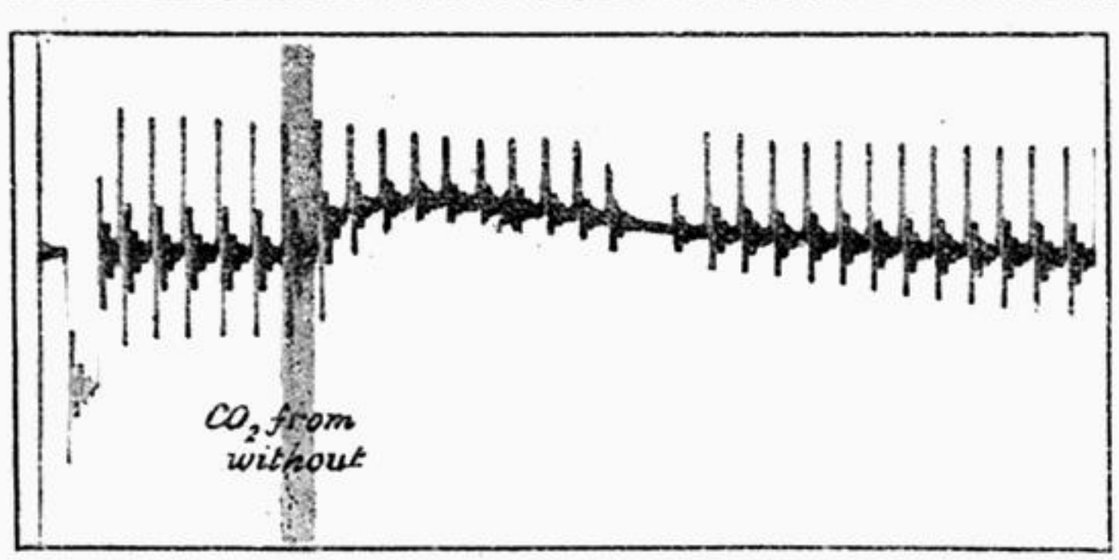
859



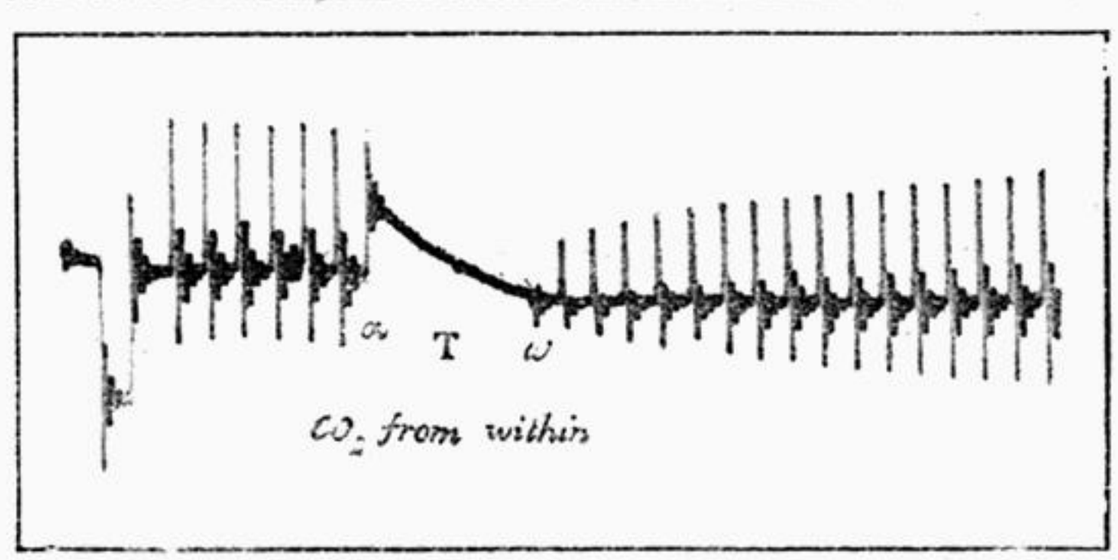
858



985



984



Summary of observations presenting typical effects of CO_2 and of tetanisation upon nerve in the three states alluded to in text.

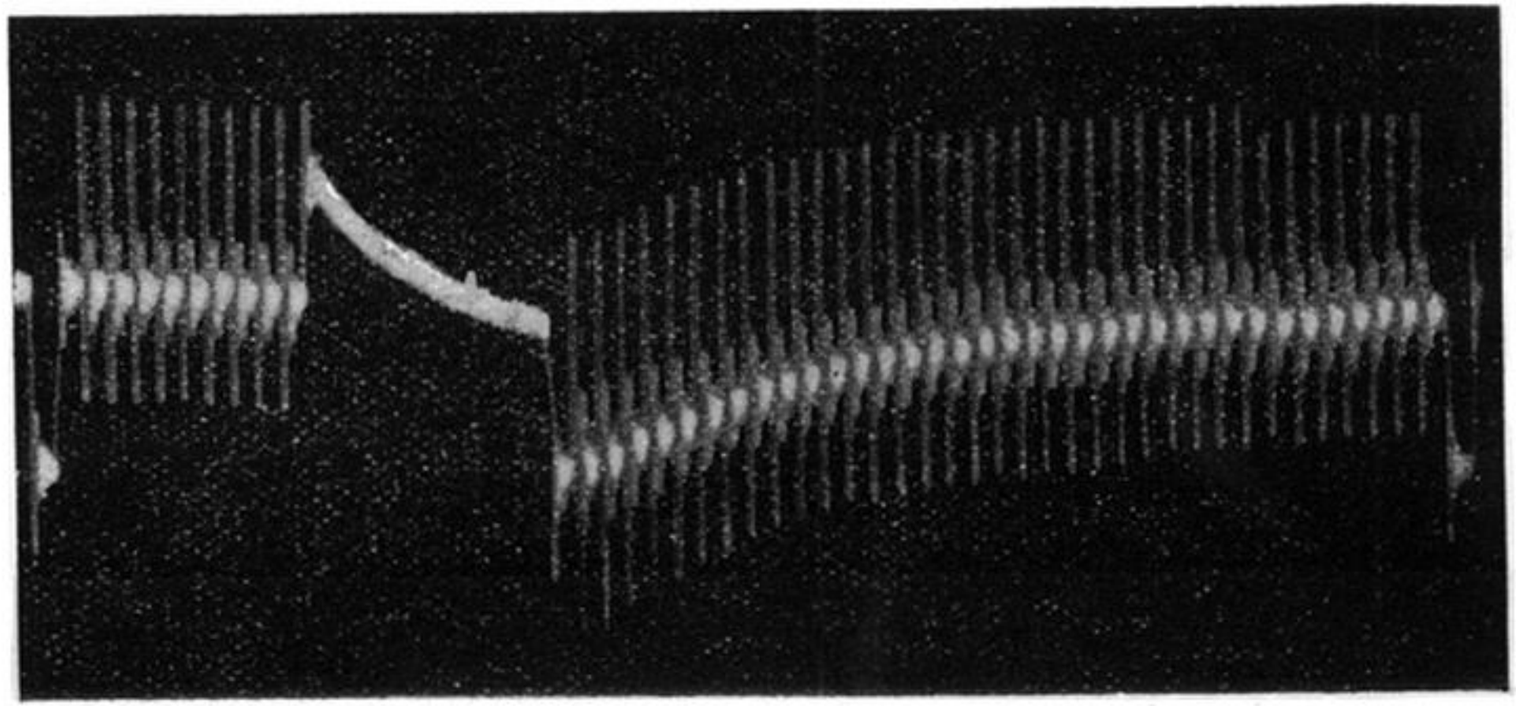
Action of CO_2 (869) and of tetanisation (675) upon nerve of the 1st stage.

,, ,, (710) ,, ,, (762) ,, ,, 2nd stage.

,, ,, (859) ,, ,, (858) ,, ,, 3rd stage, "clear effect."

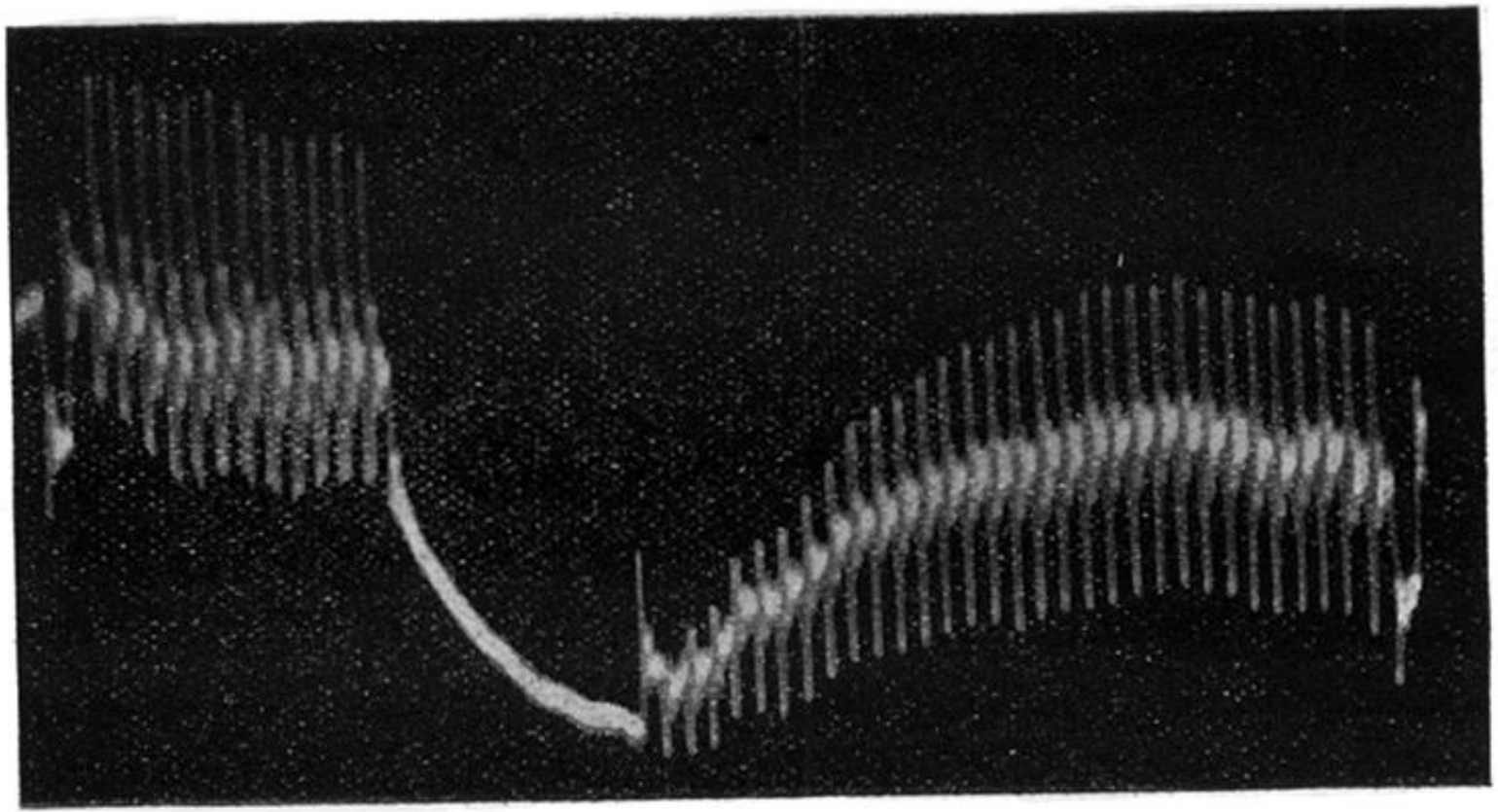
,, ,, (985) ,, ,, (984) ,, ,, ,, "disguised effect."

Fig. 36. (Obs. 905.)



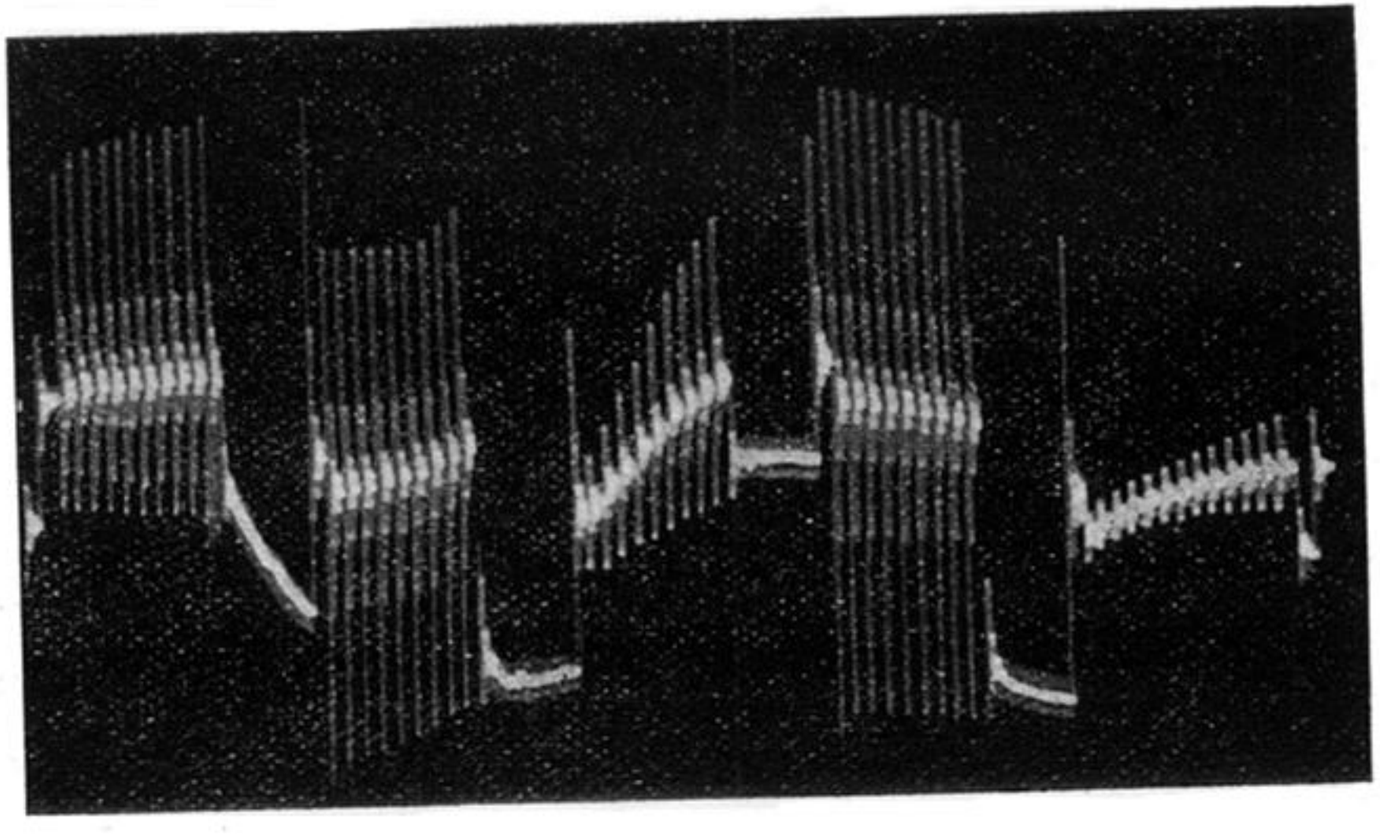
Nerve of the third stage. Exceptional effect of prolonged tetanisation. Augmentation of the positive deflection.

Fig. 37. (Obs. 912.)



Nerve of the second stage. Exceptional effect of prolonged tetanisation. Diminution of the negative deflection.

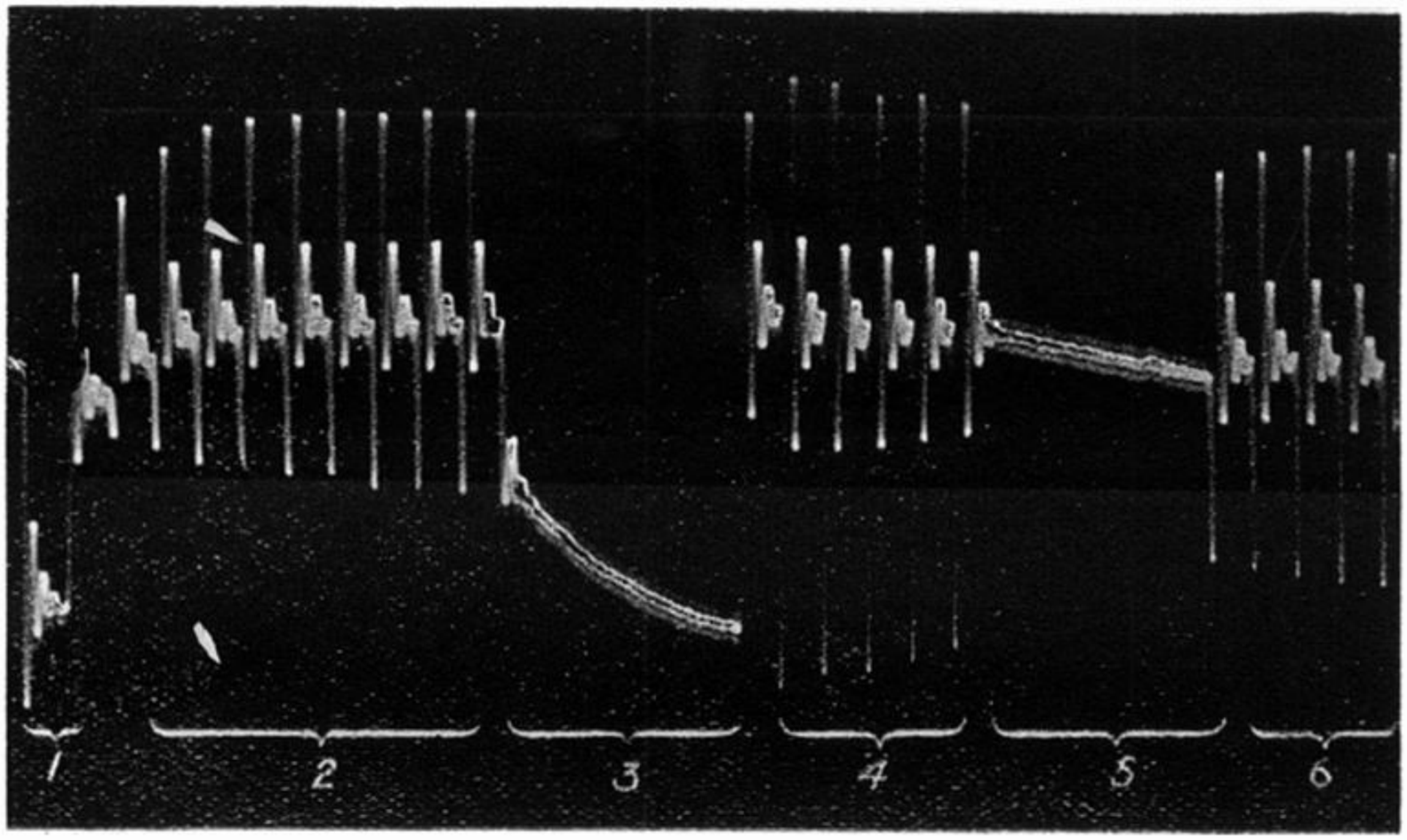
Fig. 38. (Obs. 2068.)



T 10↑ T 100↑ T 10↓ T 100↓

Nerve of the second stage. Augmented negative effect in consequence of weak tetanisation (10 units, both directions of coil). Diminished negative effect in consequence of strong tetanisation (100 units, both directions of coil).

Fig. 39. (958.)

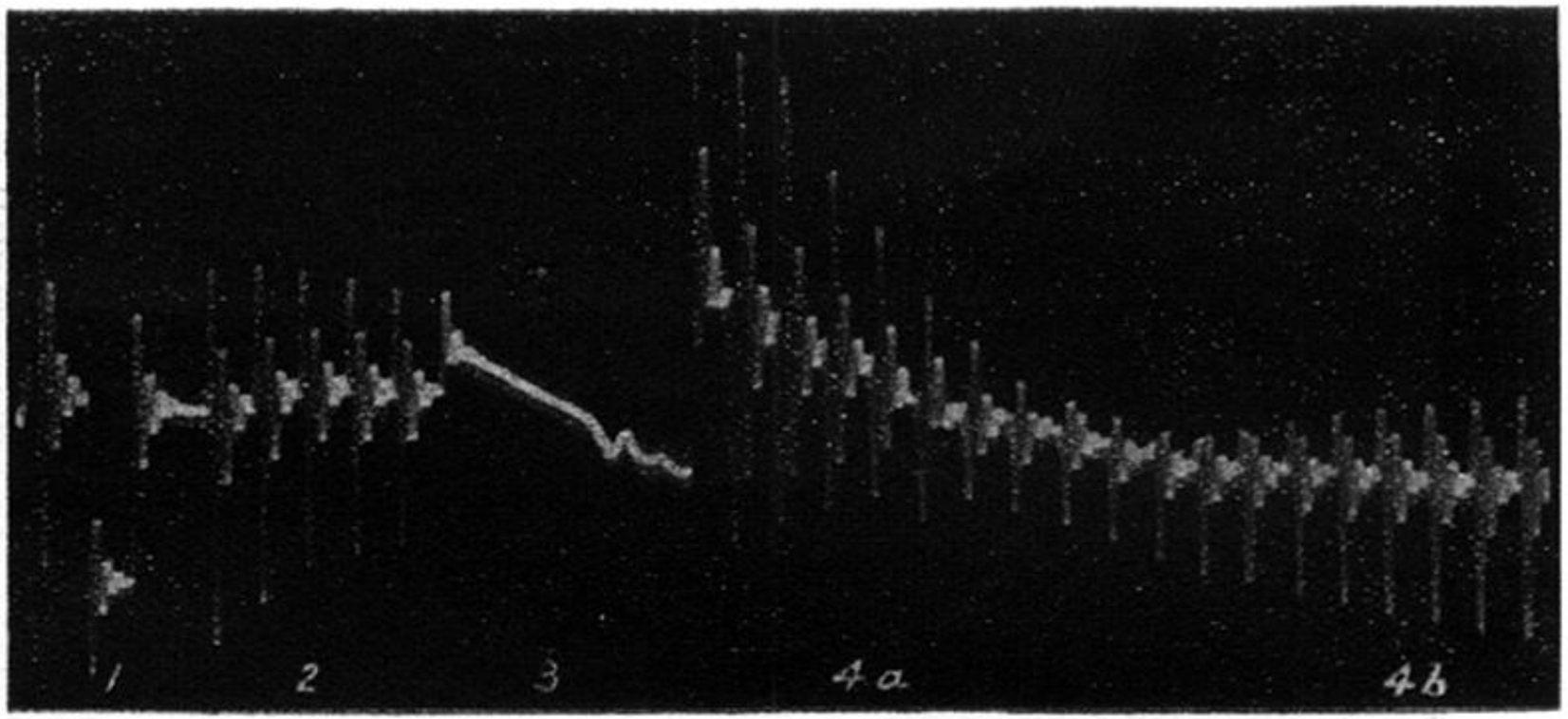


Nerve 5 hours after excision giving a deflection of the second stage. Excitation once a minute for $\frac{1}{8}$ minute with coil at 10 units. Tetanisation for 5 minutes from α to ω.

Before the tetanus the successive negative variations increase slightly forming a "staircase." After the tetanus the negative variations are increased, gradually diminishing. An interruption (*int.*) of all excitat ion shows how the diminution was delayed by the excitation itself.

		Presumable Change of CO_2.
1	Deflection by 0·001 volt	
2	Normal series at minute intervals exhibiting staircase increase	Evolution in small quantities at each tetanus.
3	Tetanus lasting 5 minutes . . .	Evolution in considerable quantity by long tetanus.
4	Normal series	Dissipation of CO_2 evolved during previous tetanus, in spite of short tetani.
5	Interruption of series by 5 minutes rest	More rapid dissipation of CO_2 effect while the latter are intermitted.
6	Normal series	

Fig. 40. (933.)

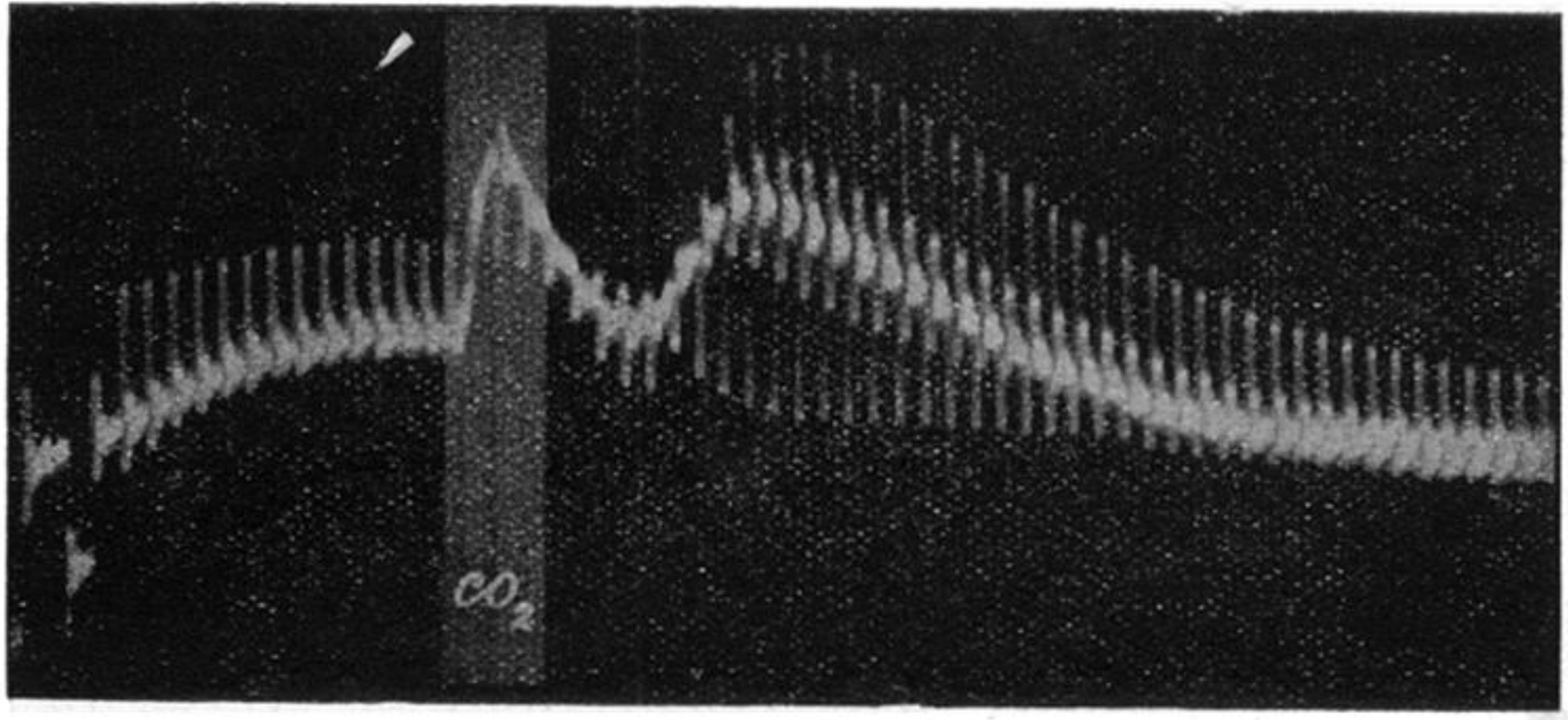


Nerve nine hours after excision, giving a deflection of the third stage, viz., N., temporarily reverted to S. in consequence of tetanisation.

1. Deflection by 0·001 volt.
2. Normal series N.S.
3. Tetanisation for 5 minutes.
4A. Normal series S.N., giving place to
4B. Normal series N.S.

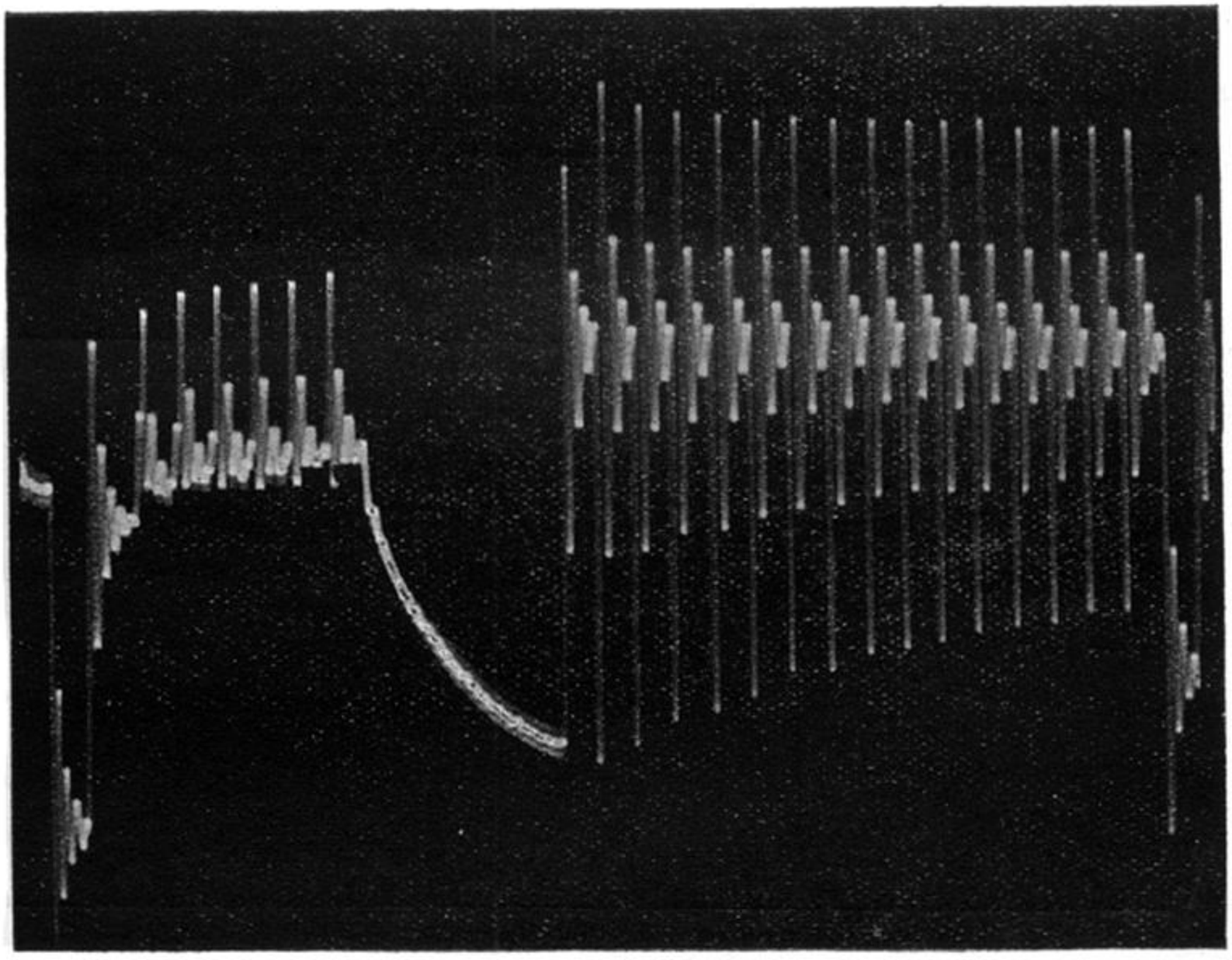
This is one of the few instances in which a negative after-deflection has been observed; the nerve had been employed two hours previously to test upon it the effect of muscle-extract.

Fig. 41. (843.)



Nerve 11 hours after excision, giving a deflection of the third stage, temporarily reversed, in consequence of CO_2 passed through the nerve-chamber for four minutes.

Fig. 42. (960.)



Nerve 10 hours after excision, giving a variation of the second stage (late) or third stage (early).

At the beginning of the record the deflection is *n.*, and the after-deflection N. With successive excitations the *n.* deflection diminishes and disappears.

With the 5 minutes' tetanus (coil at 10 units) from α to ω, there is no α deflection, a rapidly increasing negativity from α to ω, and a large positive ω deflection. Subsequently the rhythmic excitation produces a large negative deflection gradually decreasing.

Fig. 43. (Plate 2148.)

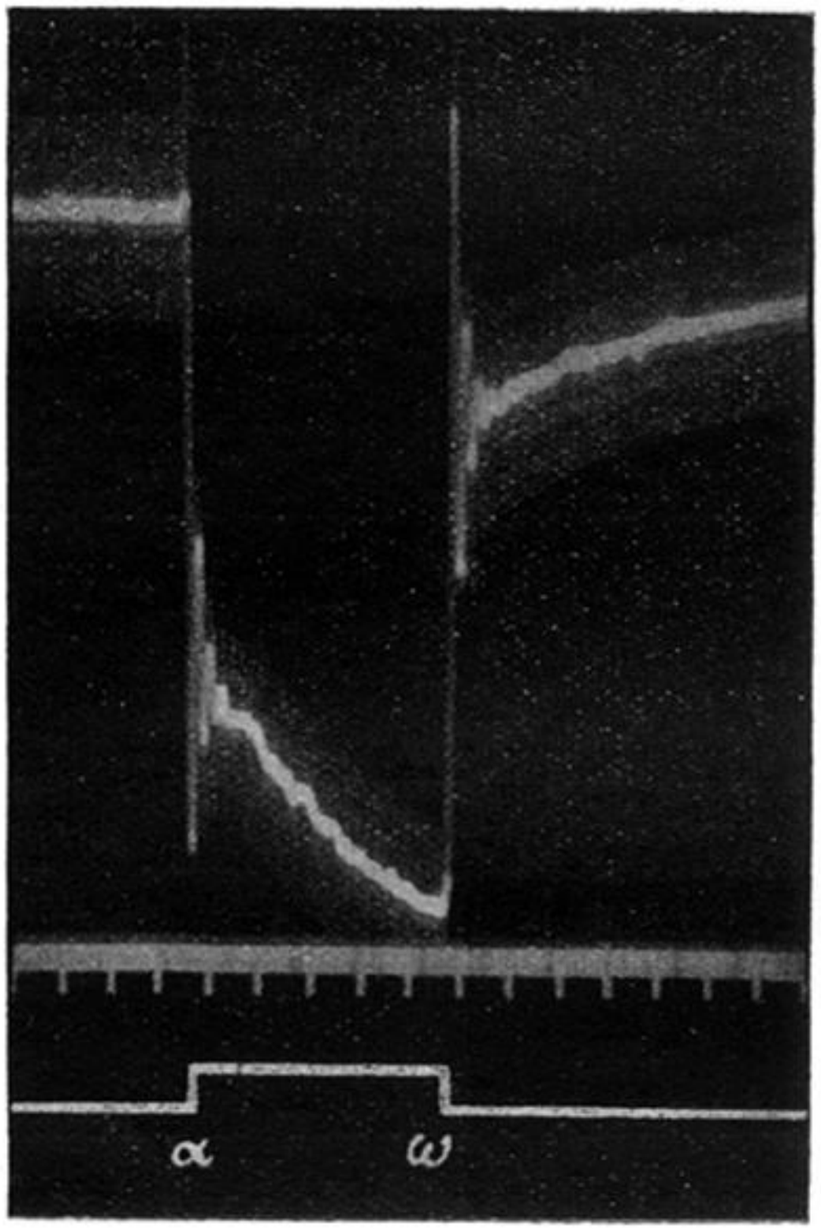


Fig. 44. (Plate 936.)

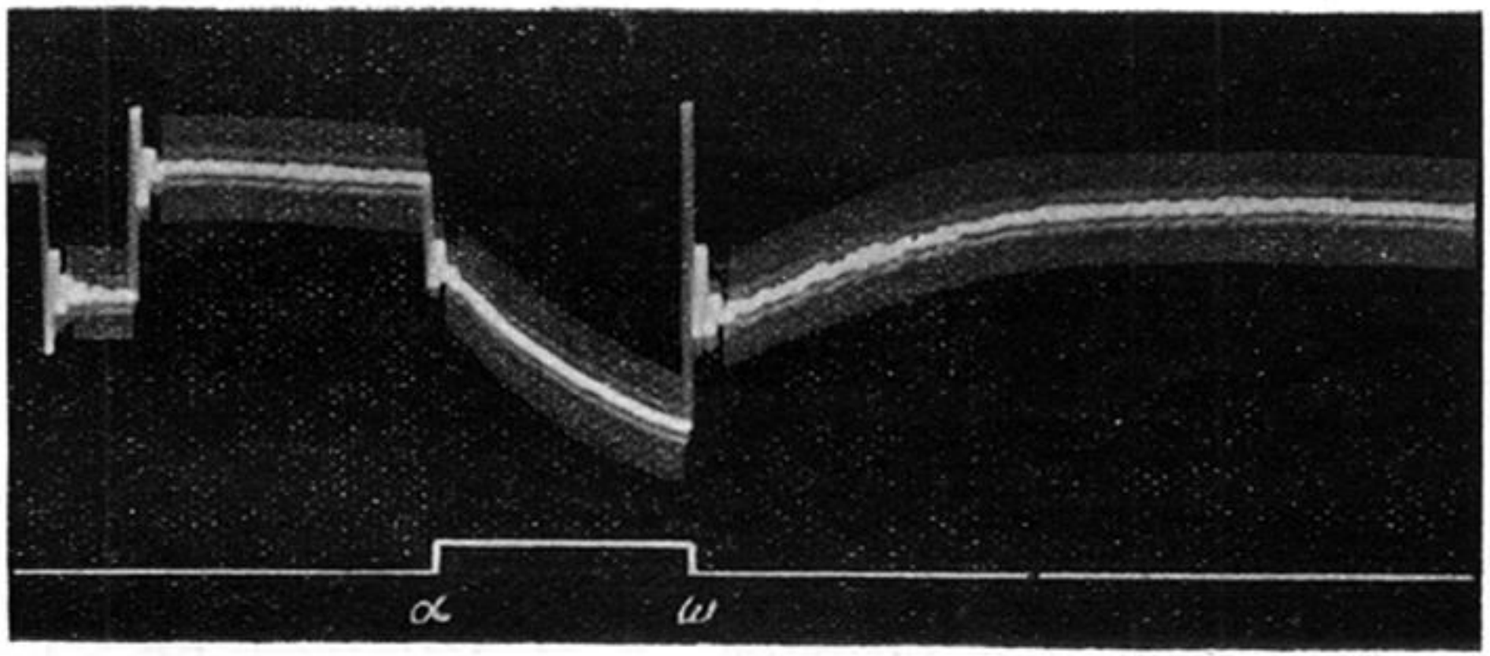
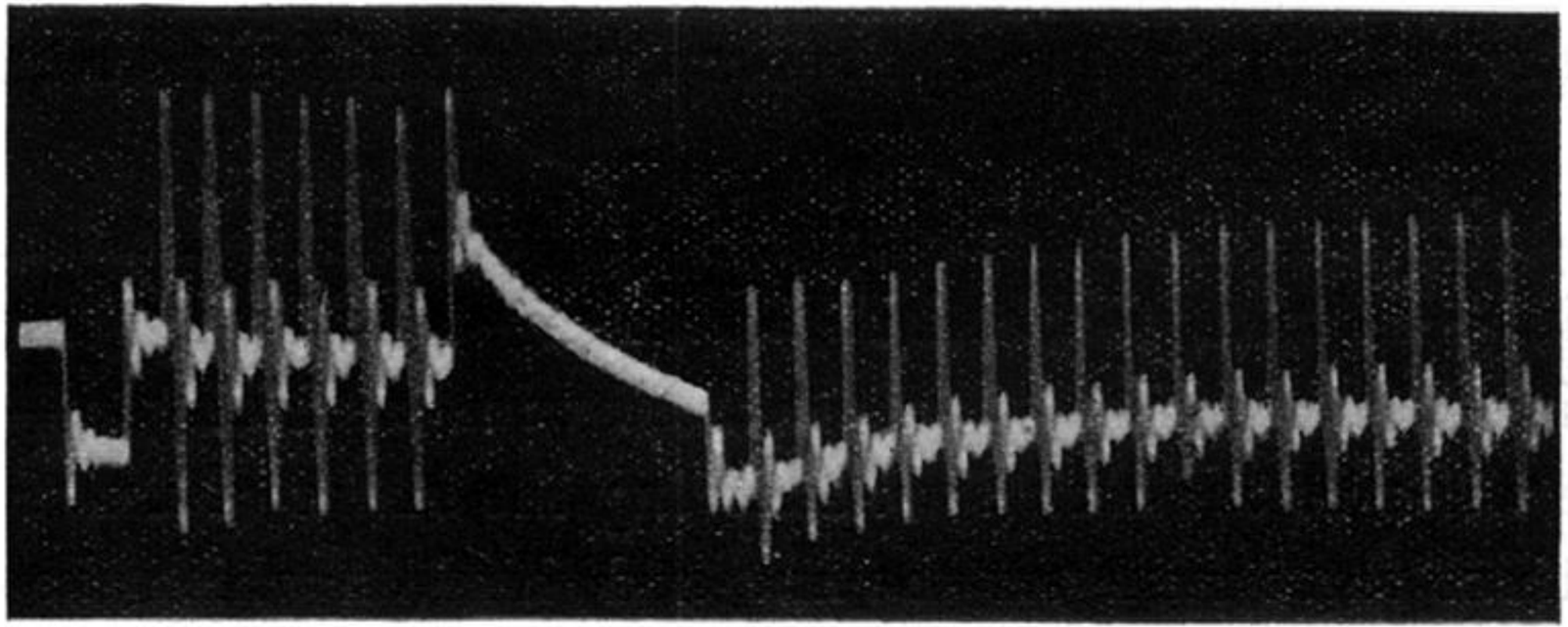


Fig. 43.—February 3, 1896.—Nerve a few minutes after excision, giving a variation of the first stage. Tetanisation for 5 minutes from α to ω; coil at 10 units.

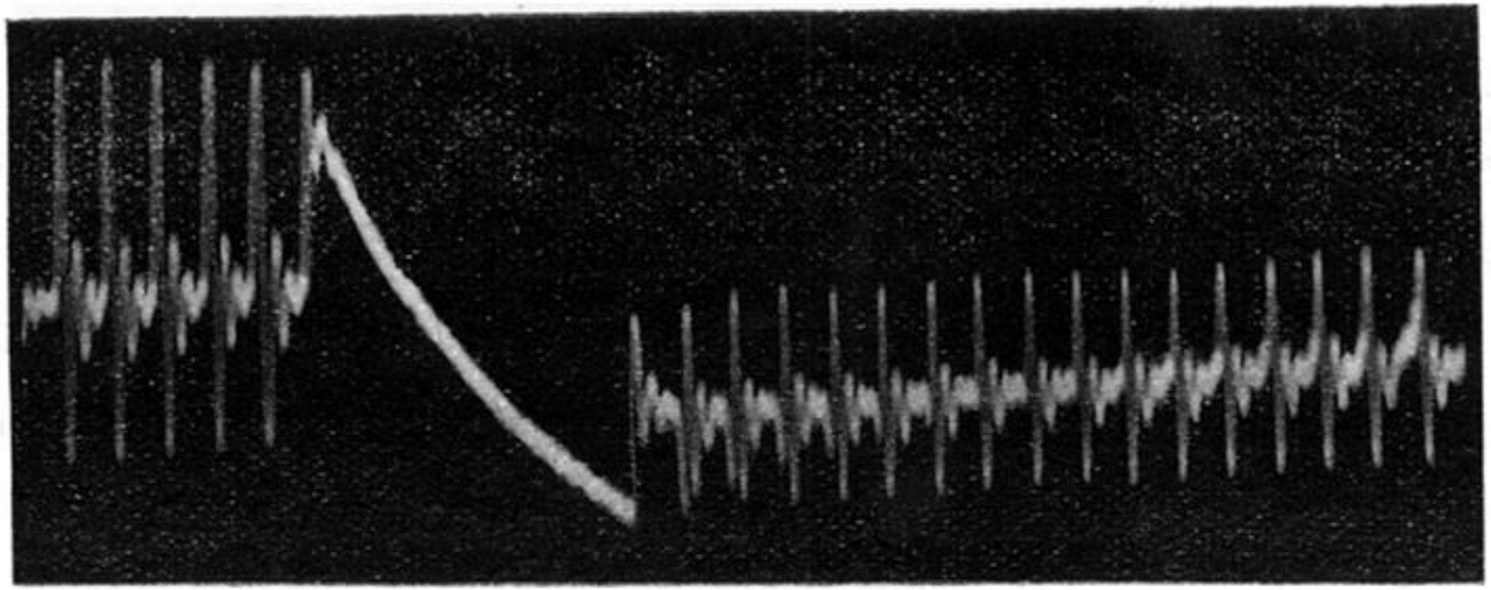
Fig. 44.—December 10, 1895.—Nerve 10 hours after excision, giving a variation of the second stage. Tetanisation for 5 minutes from α to ω; coil at 10 units.

Fig. 45. (874.)



November 30, 1895.—Nerve 10 hours after excision (previously used for observations with ether and with CO_2), giving a variation of the 3rd stage (positive), with a negative ω deflection at the end of 5 minutes' tetanisation, and a diminution of the positive effect of excitation. ("Disguised" effect.)

Fig. 46. (898.)



December 5, 1895.—Nerve several (?) hours after excision (previously used for observations with sarcosin), giving a variation of the 3rd stage, with a positive ω deflection at the end of 5 minutes' tetanisation, and a reversal of the effect of excitation. ("Clear" effect.)

Fig. 47. (826.)

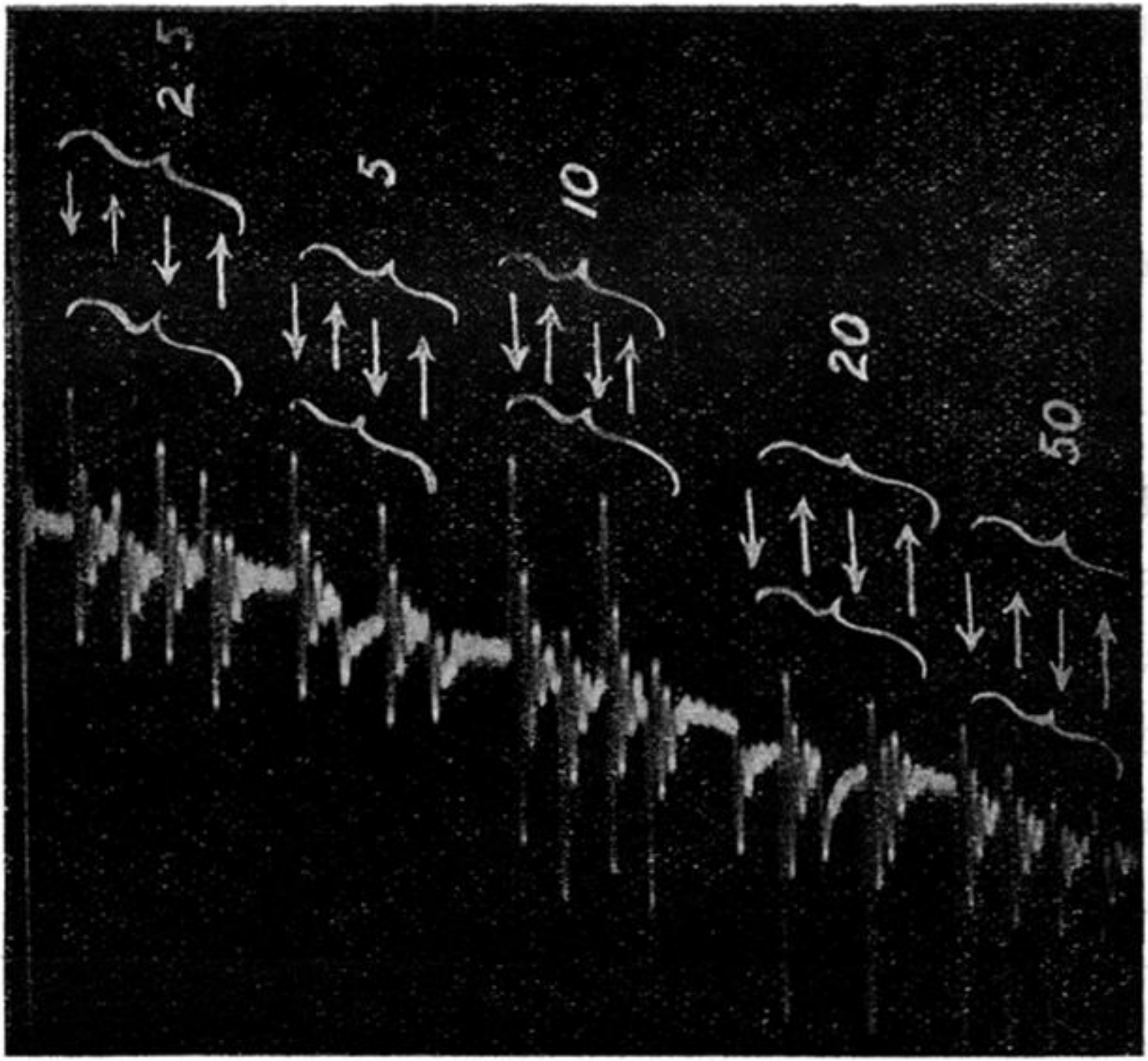


Fig. 49.

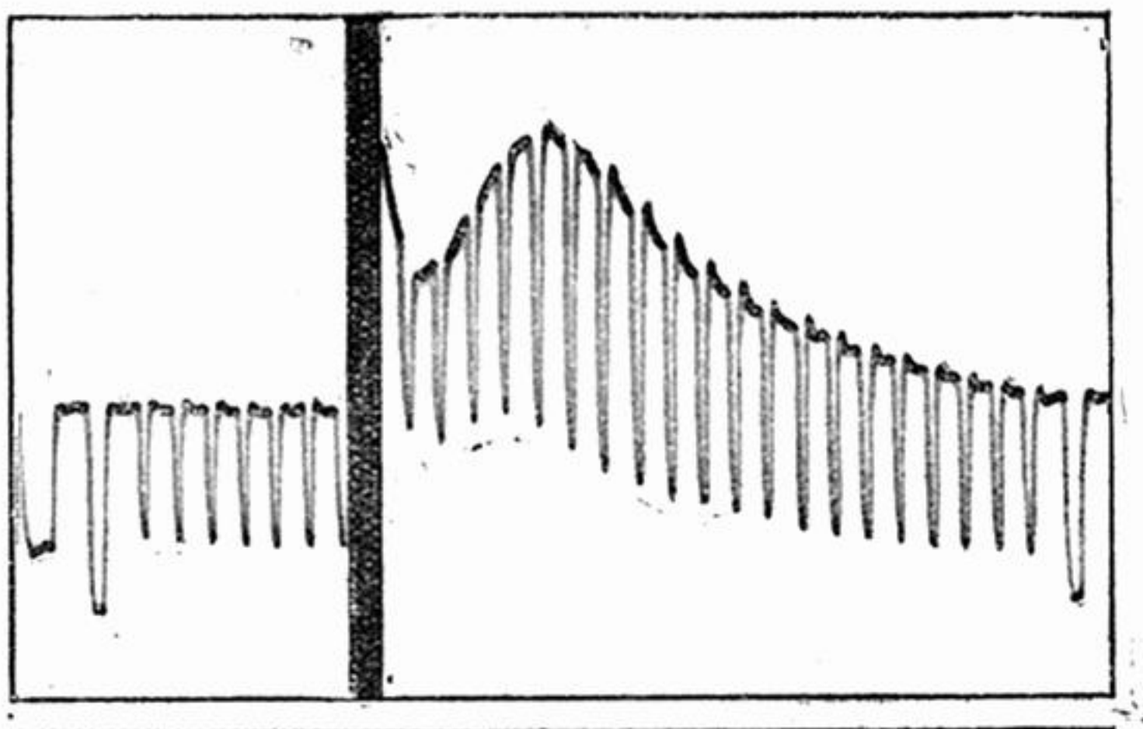


Fig. 50.

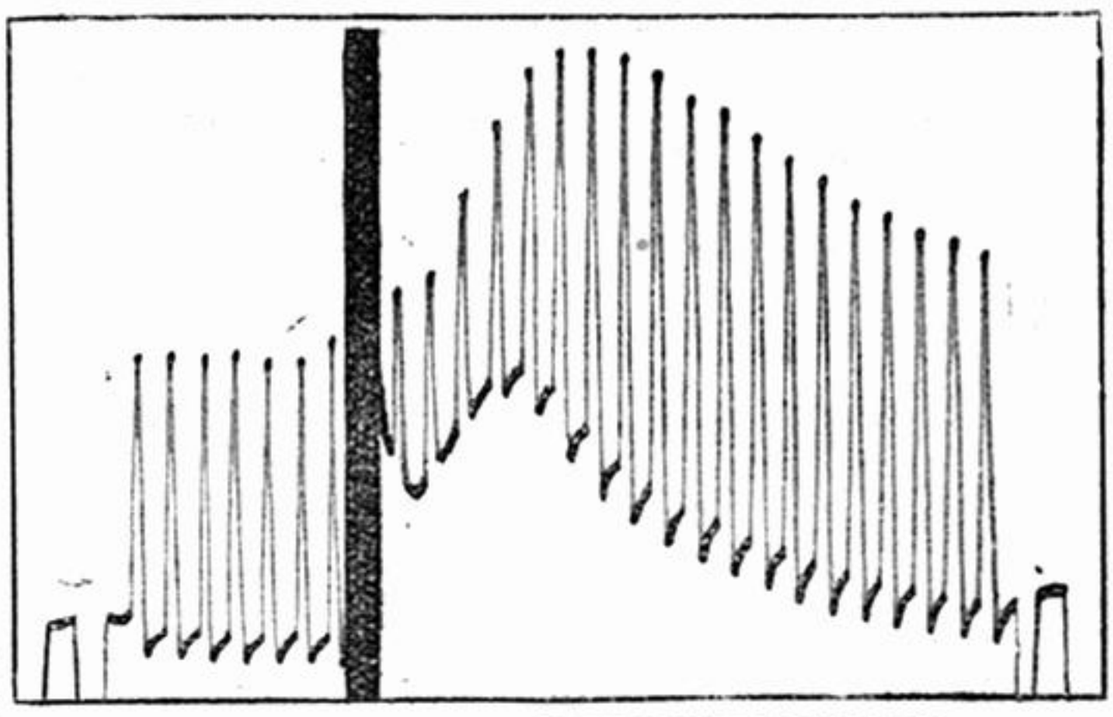


Fig. 51.

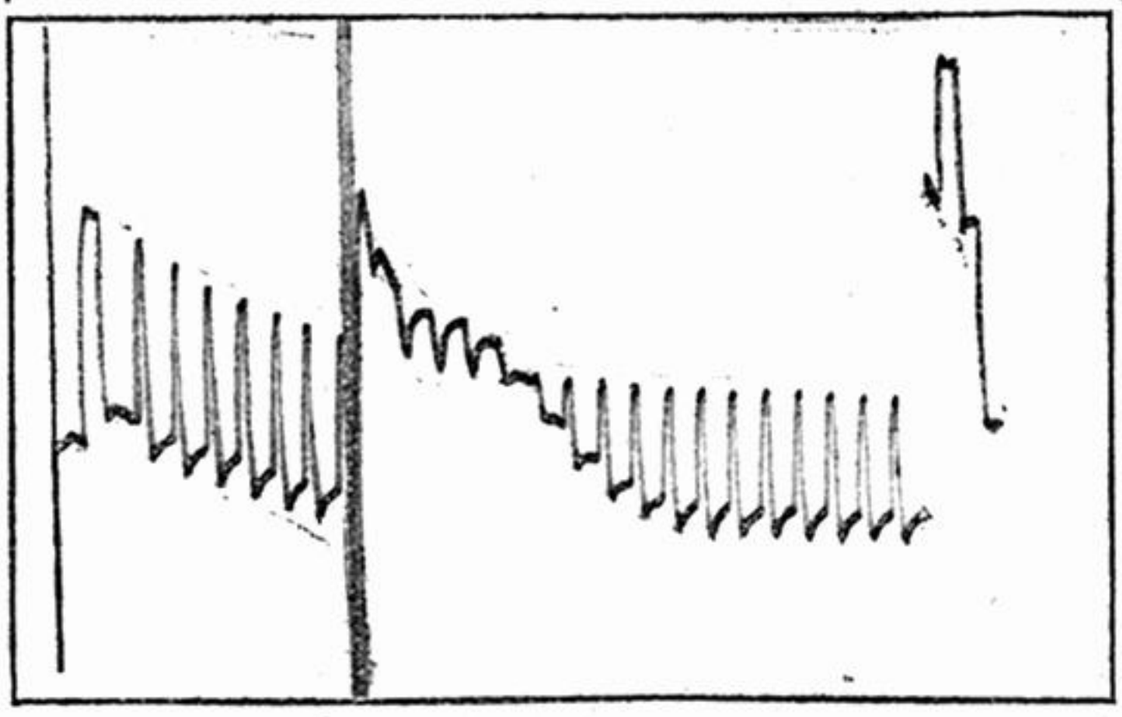


Fig. 52.

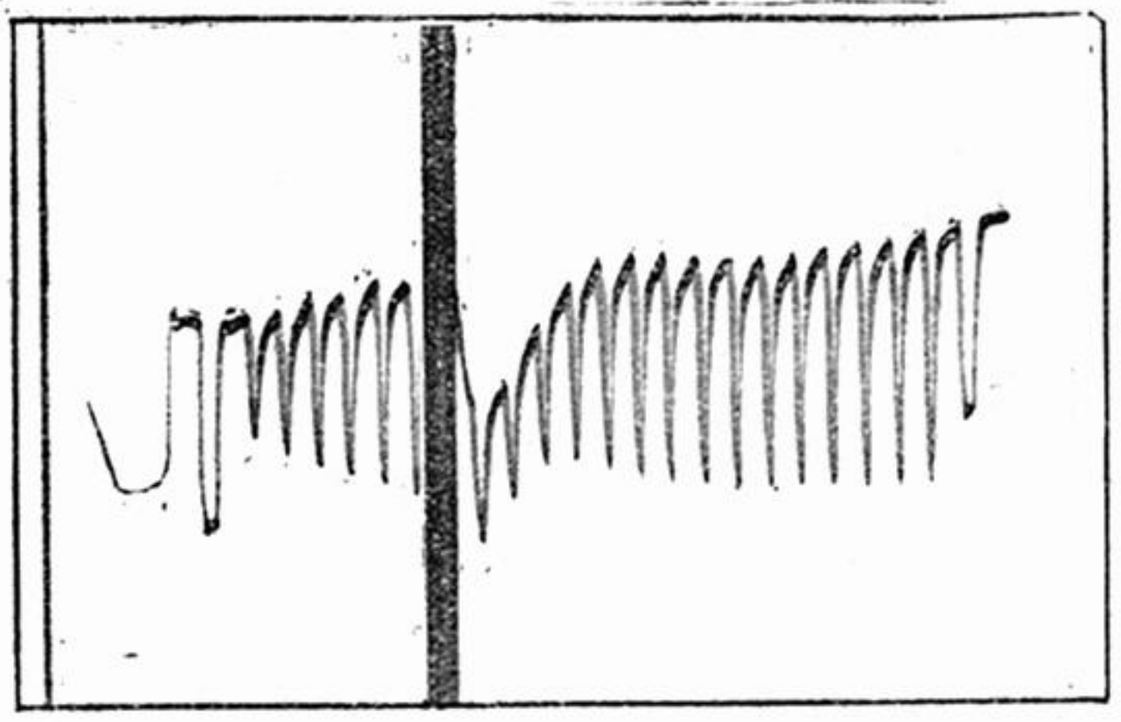


Fig. 53.

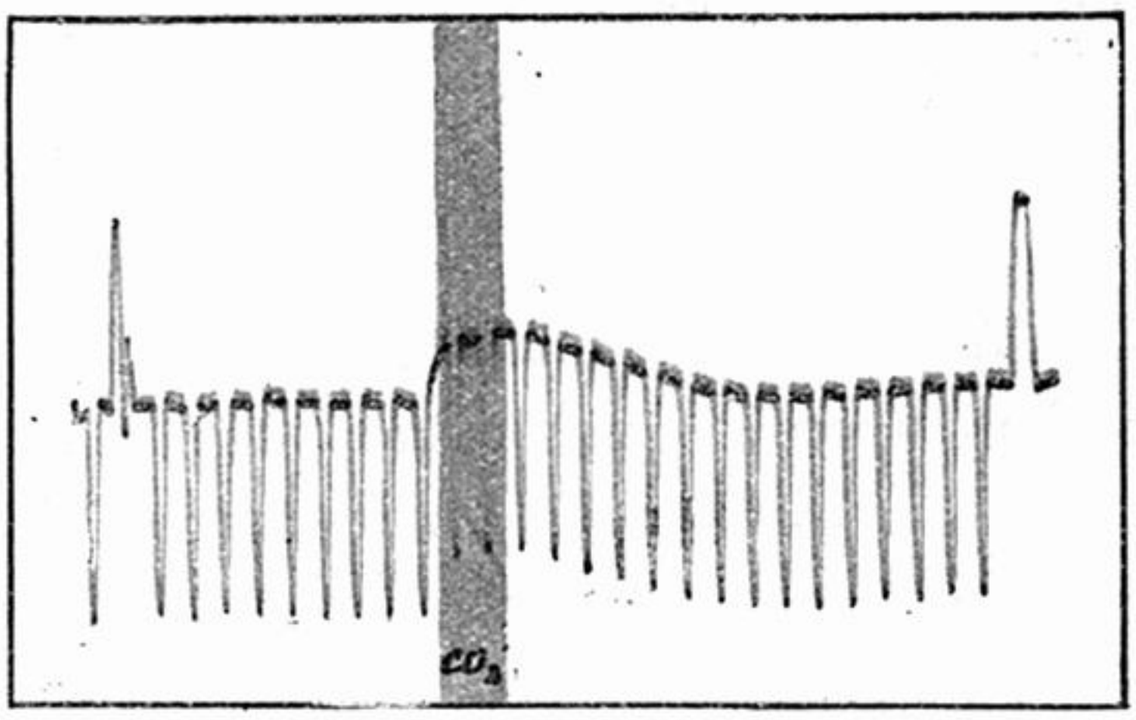


Fig. 54.

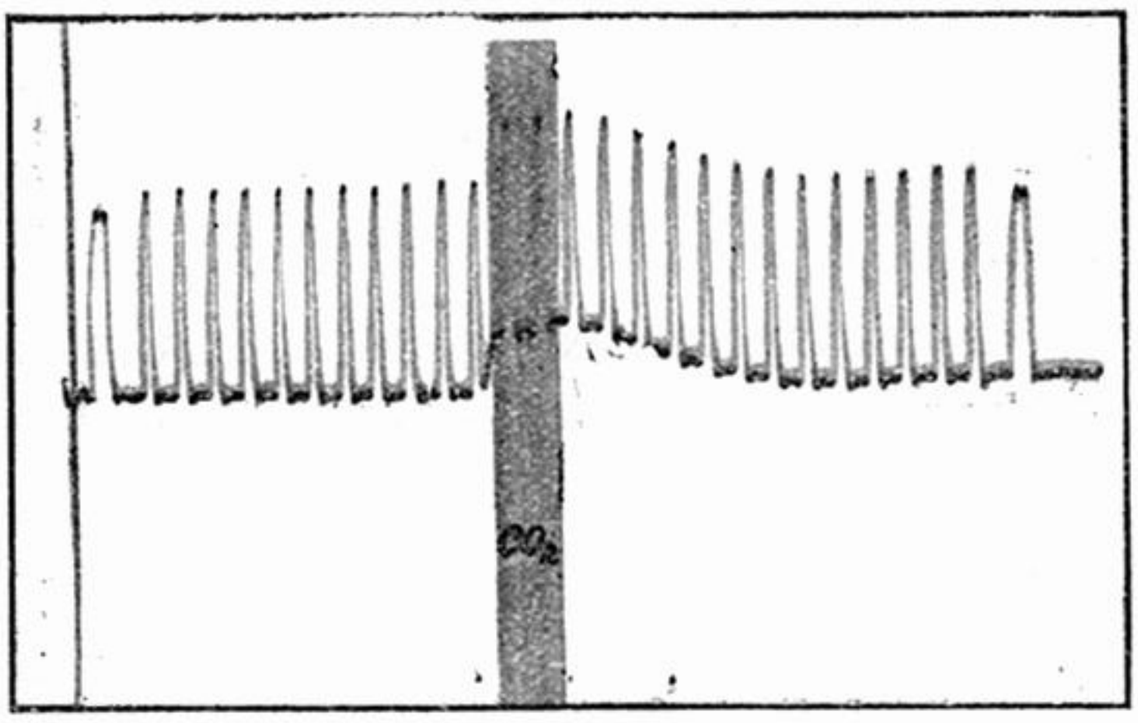


Fig. 55.

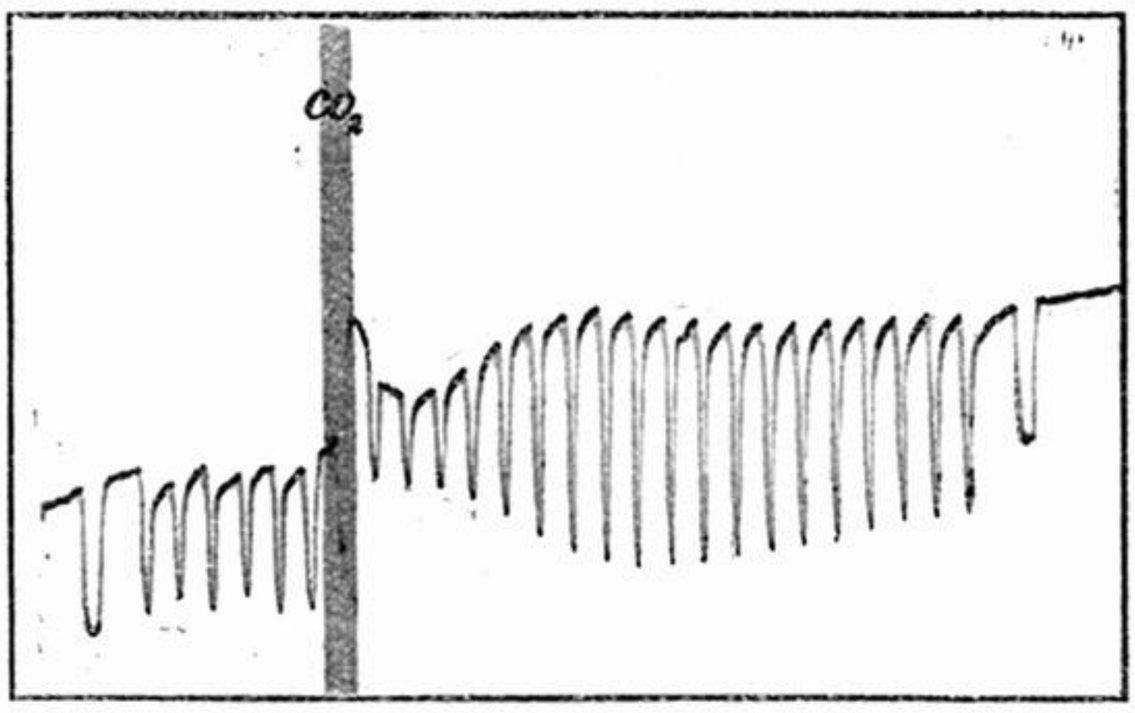


Fig. 56.

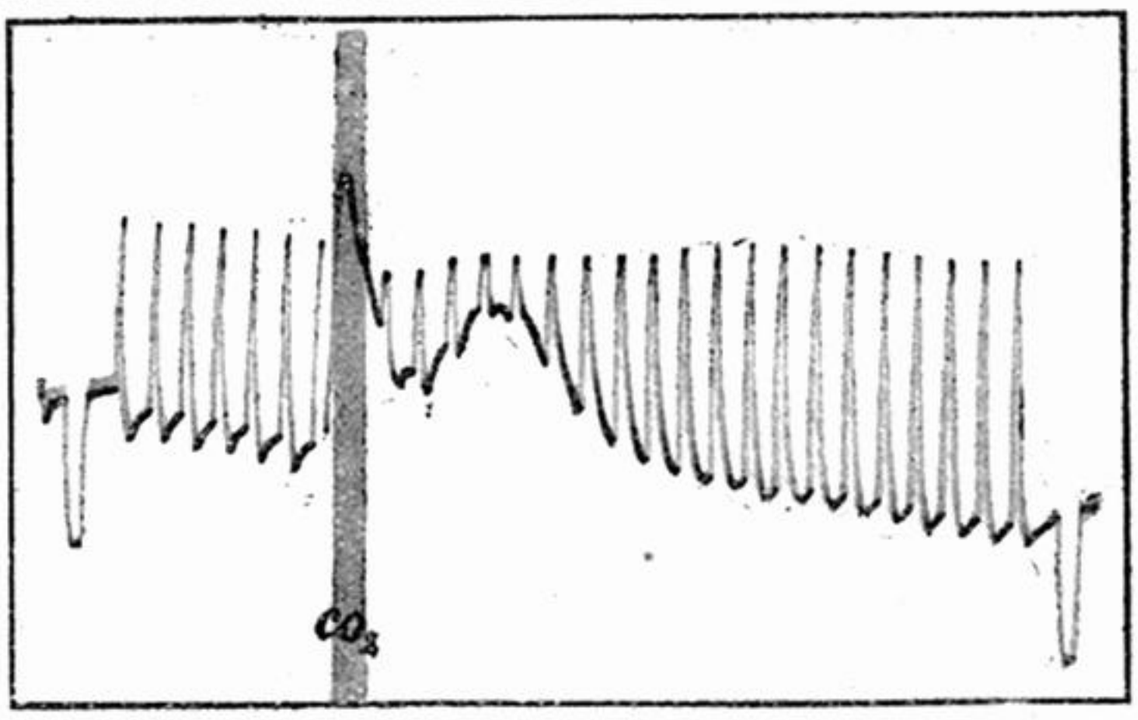


Fig.	Plate.		Fig.	Plate.	
49	2195	Effect of CO_2 on katelectrotonus	50	2199	Effect of CO_2 on anelectrotonus
51	2181	„ „ a katelectrotonic decrement	52	2185	„ „ an anelectrotonic decrement
53	2207	„ „ a S. polarising current	54	2208	„ „ a N. polarising current
55	2204	„ „ a S. polarisation increment	56	2203	„ „ a N. polarisation increment

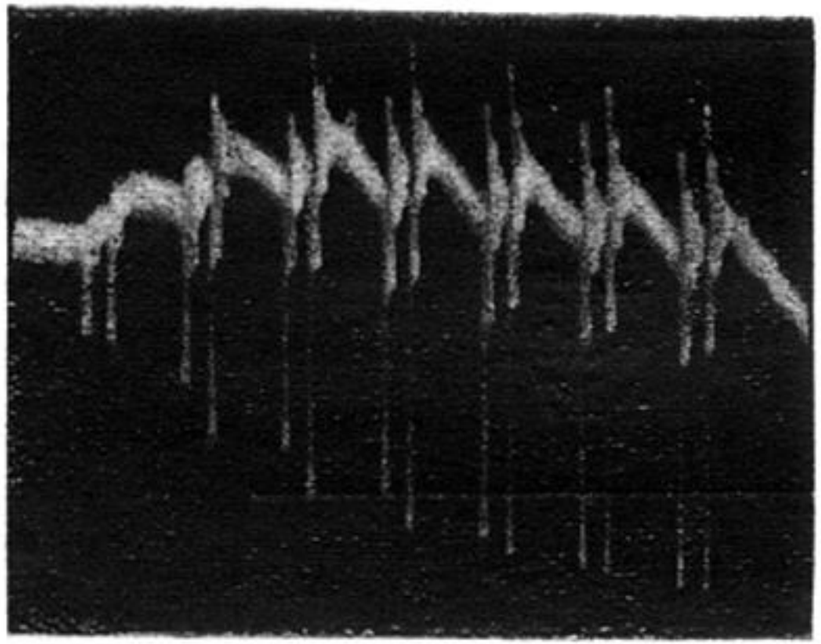
The above eight records were taken by the dead-beat galvanometer, with connections as indicated in fig. 48.

In the observations on katelectrotonus and anelectrotonus (figs. 49 and 50), the polarising current was by one Leclanché cell (= 1·4 volt) with distances (*a*) between polarising electrodes, (*b*) between polarising and galvanometer electrodes, (*c*) between galvanometer electrodes, each = 10 millims. An interrupter (make = 15 secs., interval = 45 secs.), was placed in the polarising circuit.

In the observations on the two electrotonic decrements (figs. 51 and 52), the polarising and exciting currents entered the nerve by the same pair of unpolarisable electrodes (figs. 48D and 48E), the interrupter was in the exciting circuit.

In observations on the polarisation increment (figs. 55 and 56), the polarising current was taken = 0·05 volt. (figs. 48B and 48C). The interrupter was in the exciting circuit.

Figs. 58, 59 (Plates 833, 836).



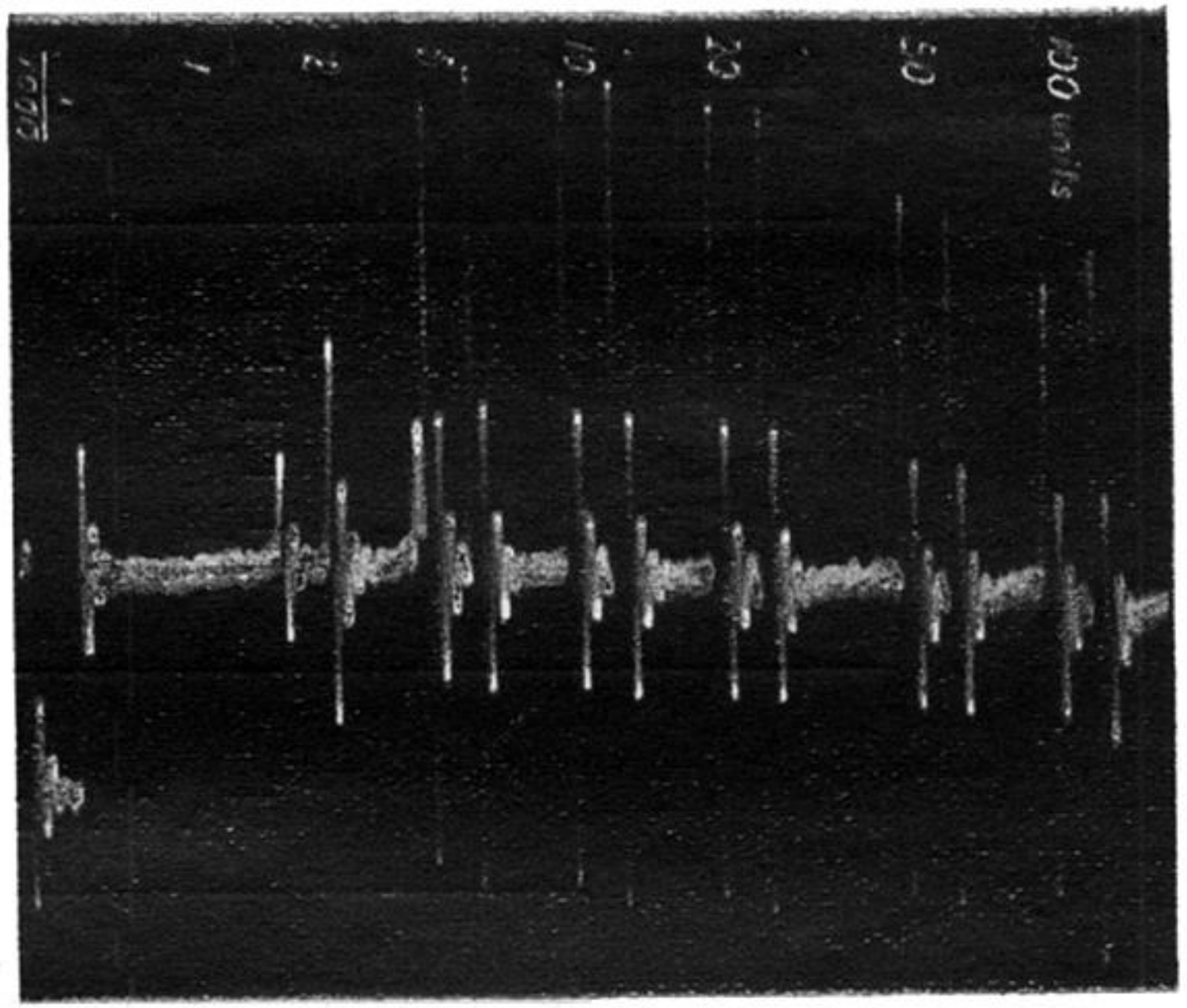
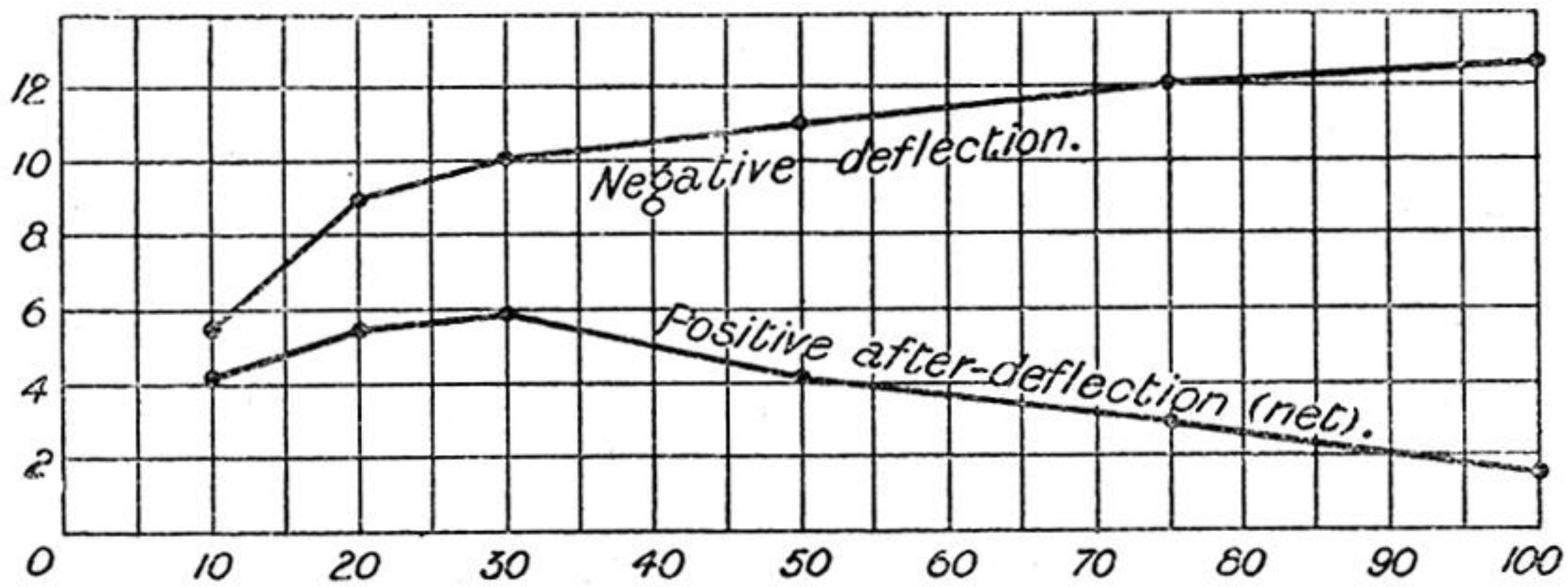


Fig. 58.—Records from which the measurements were taken for the construction of the curves of fig. 57.

Fig. 59.—Excitations in pairs, first with break descending, *i.e.*, towards the galvanometer electrodes, then with break ascending. Only the former have been used for the construction of the curves.

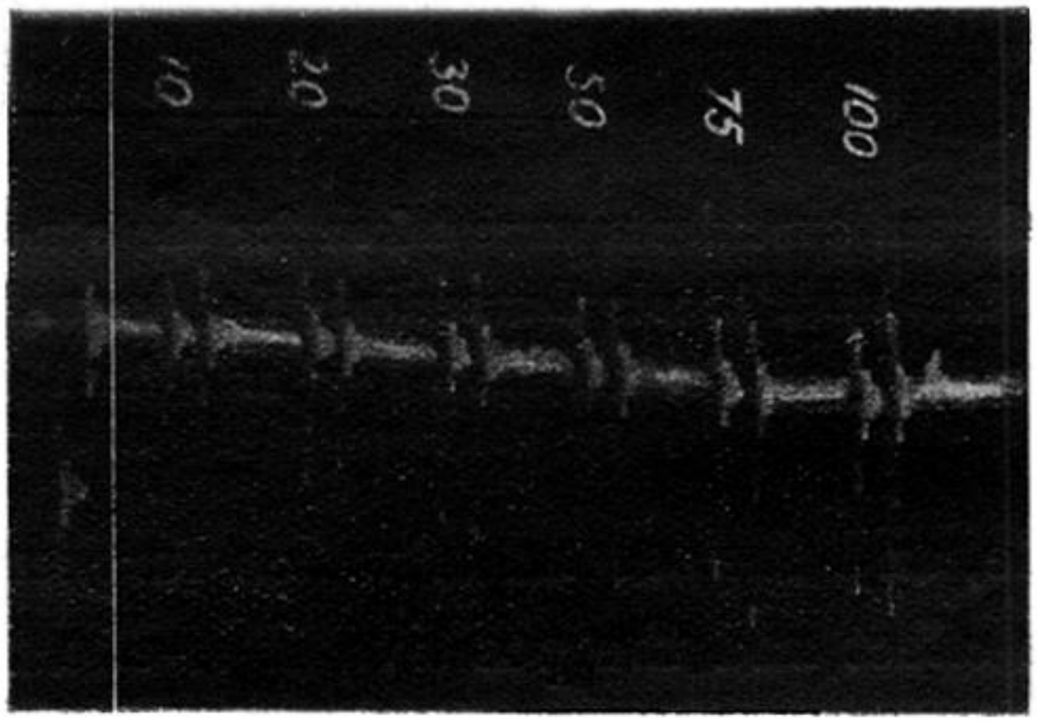
Fig. 60.



2070

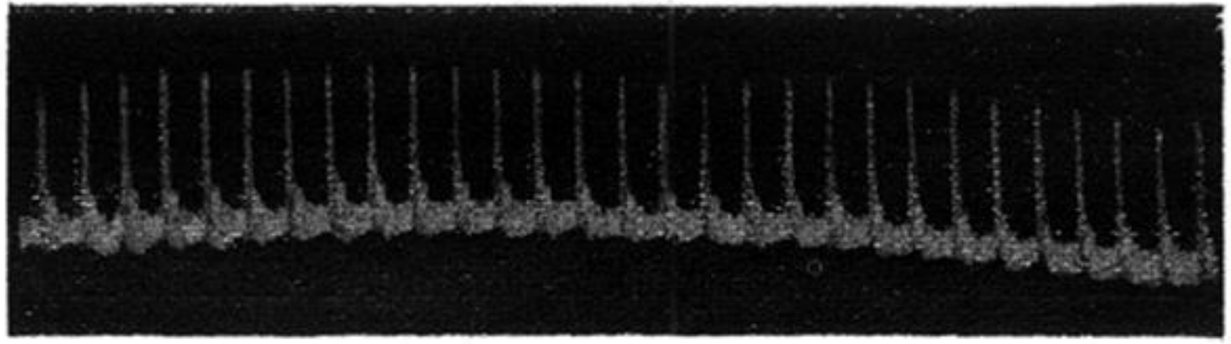
Strength of stimulation.	Magnitude of negative deflection.	Magnitude of positive after-deflection.*
10	− 5·5	+4·3
20	− 9	+5·5
30	−10	+5·8
50	−11	+4·2
75	−12	+3·0
100	−12·5	+1·7

Fig. 61. 2070.



Record from which the measurements were taken for the construction of the curves in preceding figure. Excitation in pairs, first with descending break, then with ascending break, only the former being used for the curves.

Fig. 62. (Plate 51.)



February 5, 1895.—Portion of a prolonged series of retinal deflections caused by stimulation of the frog's eyeball at intervals of one minute. Each stimulation consists in the light of a standard candle at a distance of two feet acting for a period af $7\frac{1}{2}$ seconds. The electromotive value of each deflection is about 0·0001 volt.

Fig. 63. Key-plate 763.

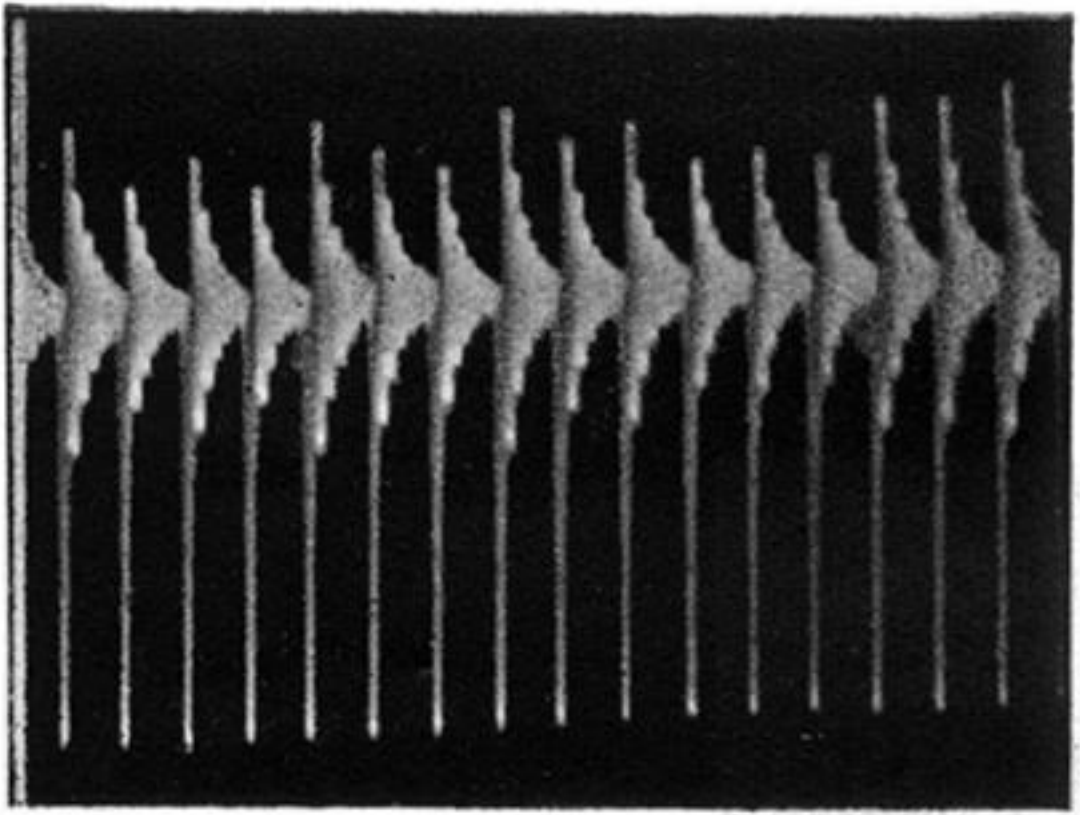


Fig 64. Key-plate 2041.

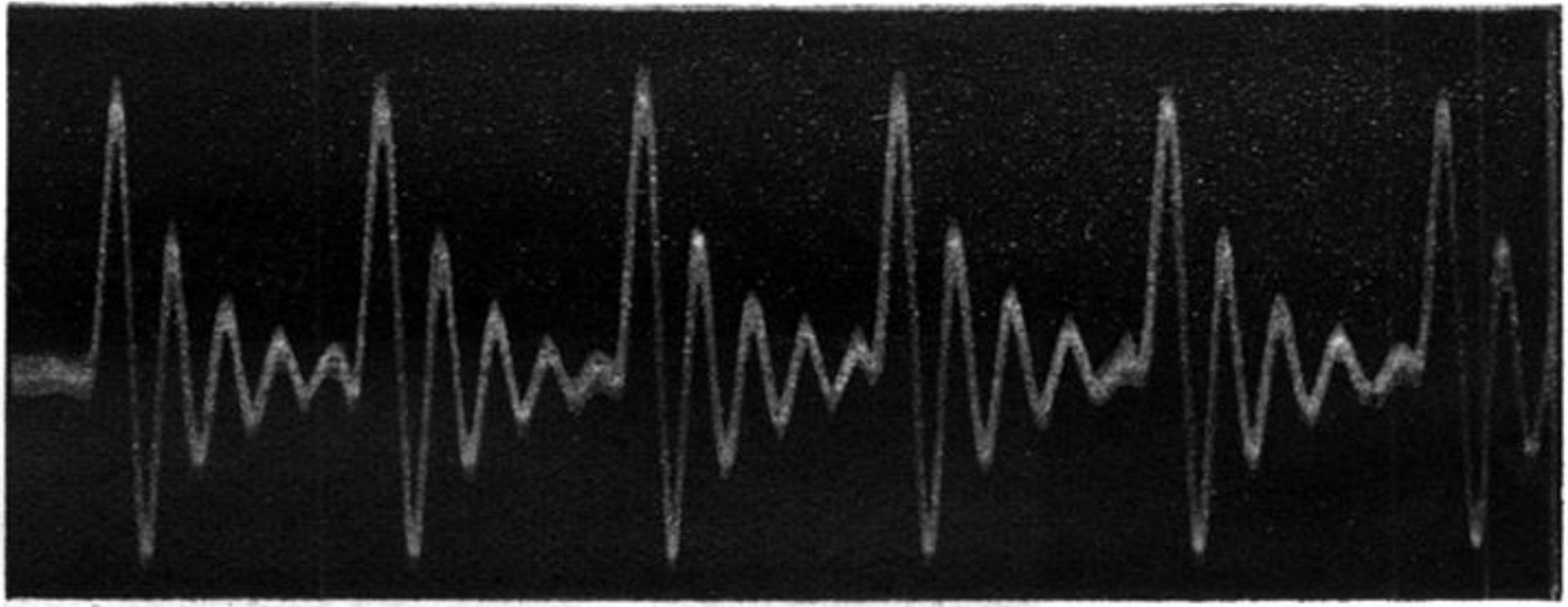


Fig. 65.

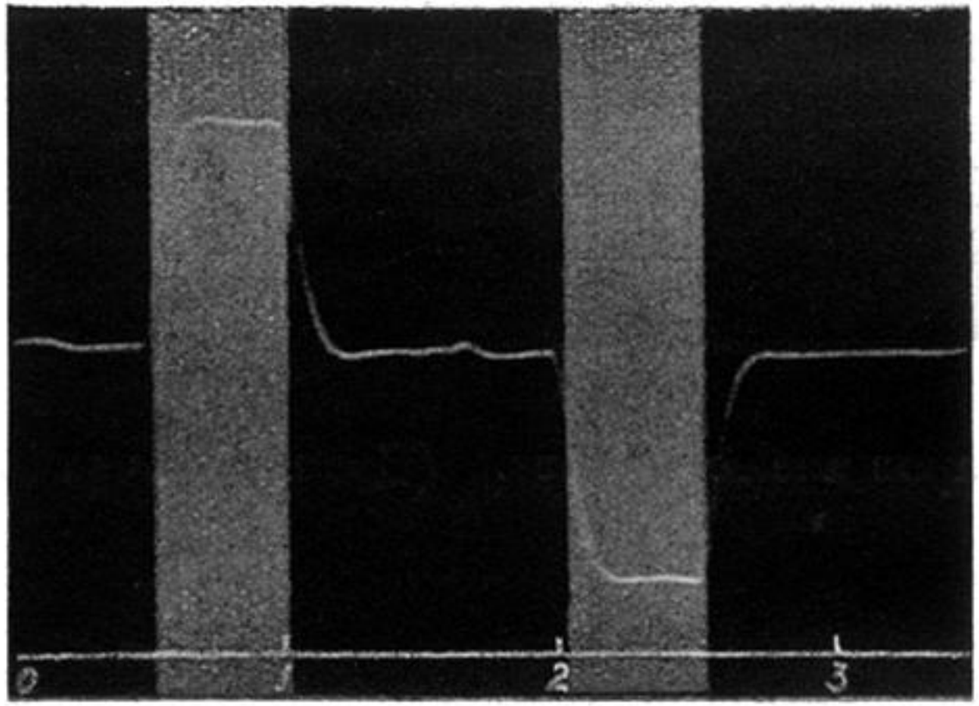


Fig. 66. (Obs. 2177.)

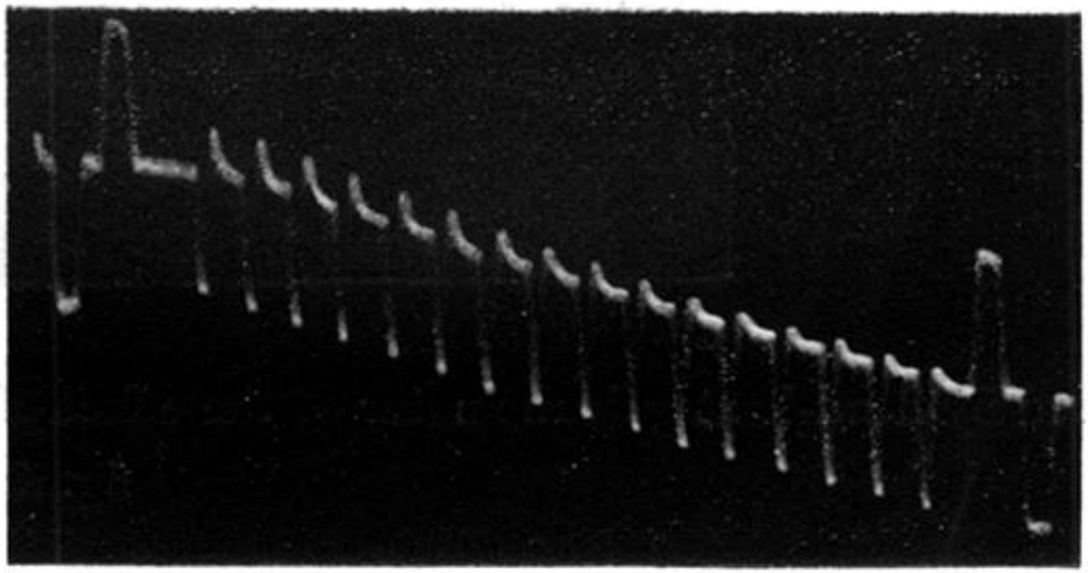
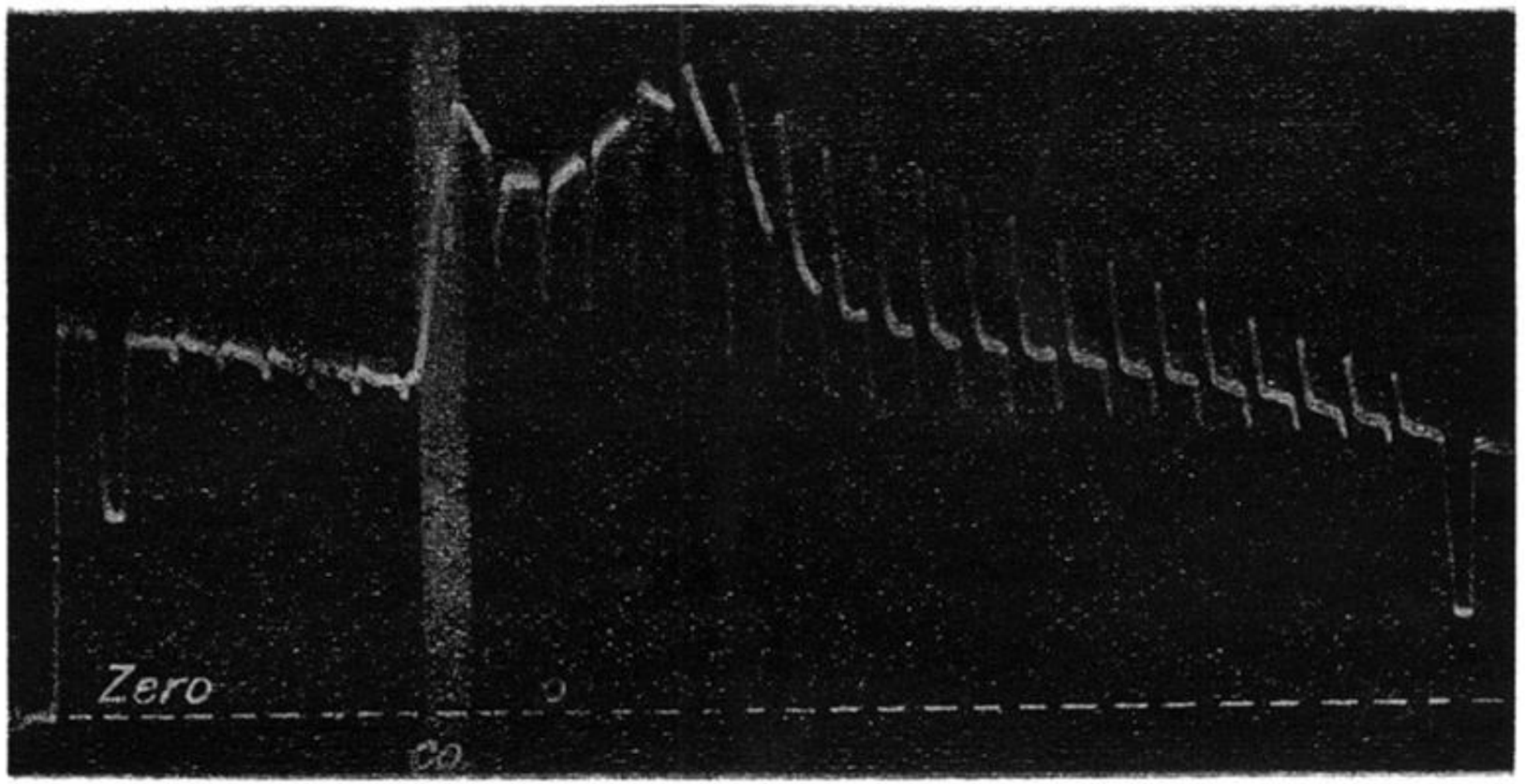


Fig. 65.—Record by the dead-beat galvanometer of an E.M.F. of 0·001 volt through a nerve, first in one, then in the opposite direction; time of closure = $\frac{1}{2}$ minute.

To show the falling time (= 10 to 12 seconds), and that the galvanometer is sufficiently dead-beat to permit us to take an excursion above the line, as in Plate 2177, to be significant of positive after-effect.

Fig. 66.—February 10, 1896.—Nerve in the late first or early second stage. To show the progressive diminution of the positive after-deflection.

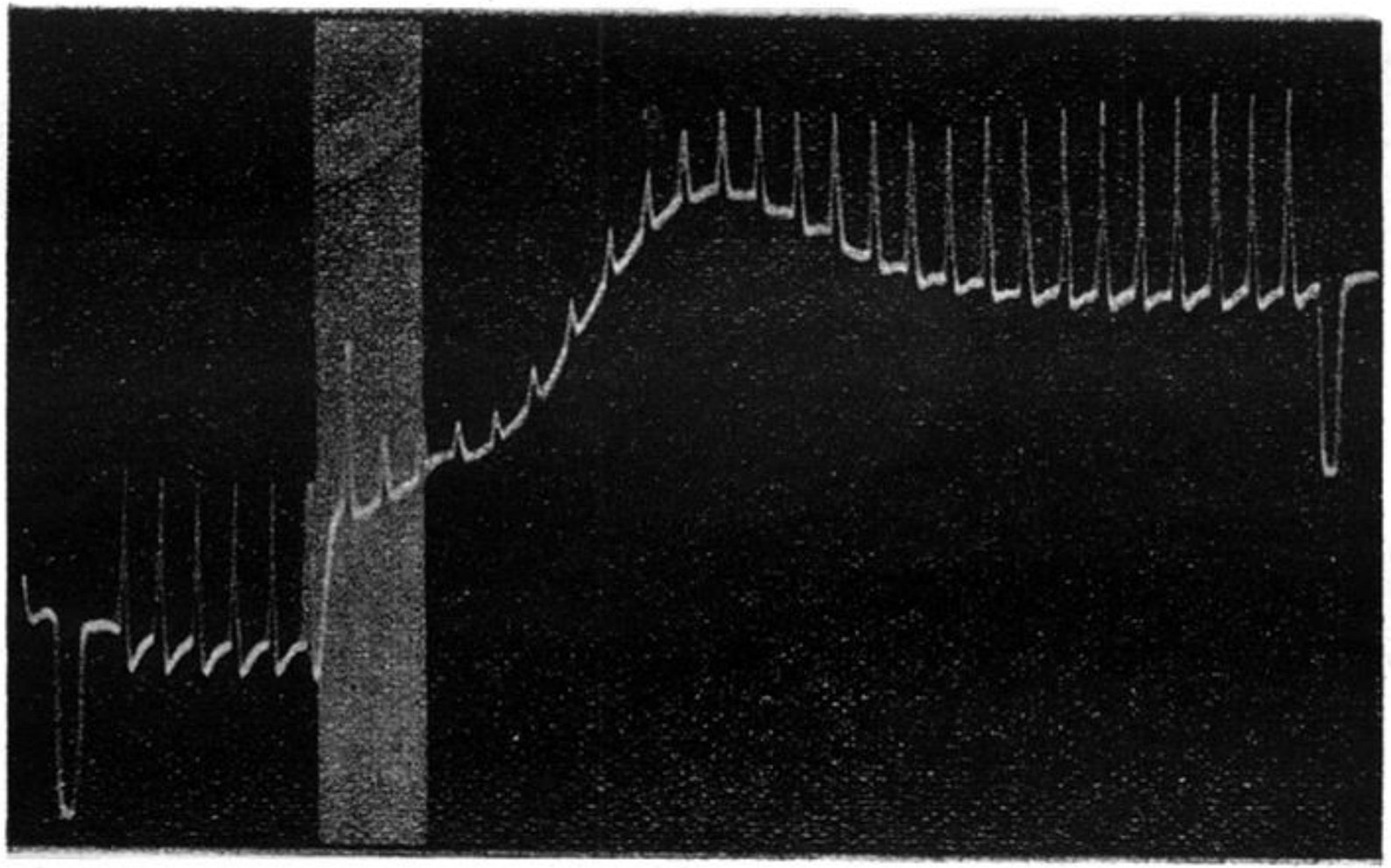
Fig. 67. (Obs. 2249.)



Record by dead-beat galvanometer of the effect of carbon dioxide on a very small negative variation (Connections as in fig. 3.)

This figure exhibits a point that has not been commented upon in the text, but is frequently witnessed in prolonged experiments, viz., a greatly augmented positive after-effect subsequent to the primary or secondary increase of the negative effect.

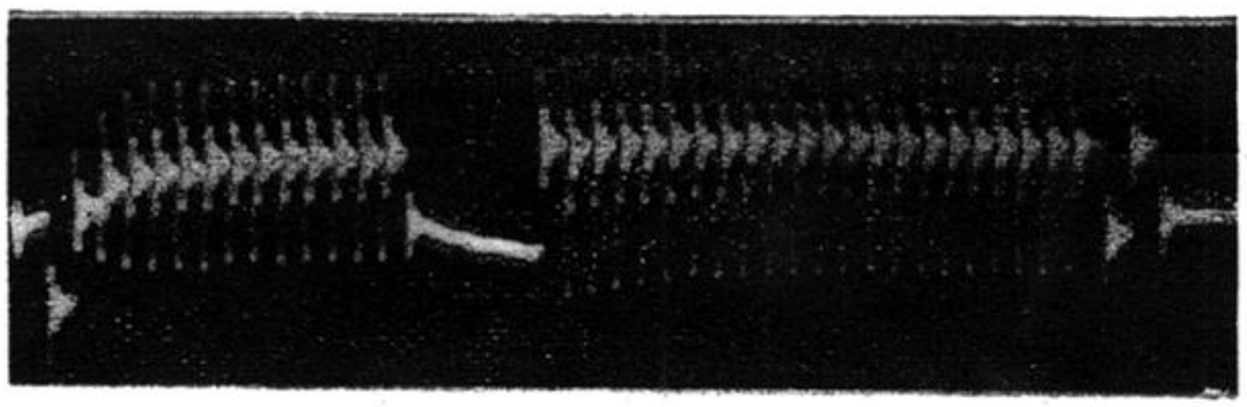
Fig. 68. (Obs. 2169.)



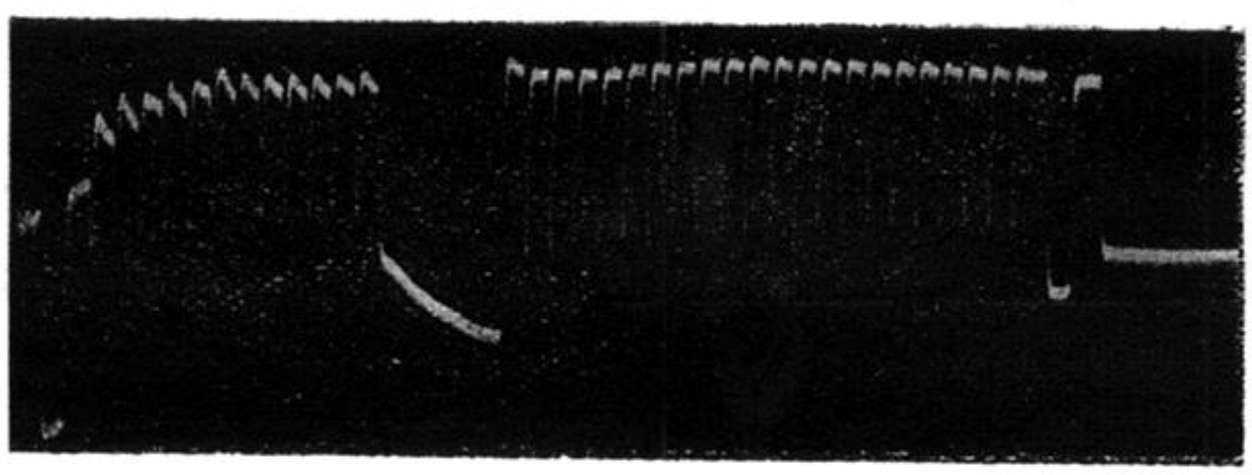
Record by dead-beat galvanometer of the effect of ether upon anelectrotonus. (Connections as in fig. 48, D.)

Fig. 69.

(3180, T.)



(3181, M.)



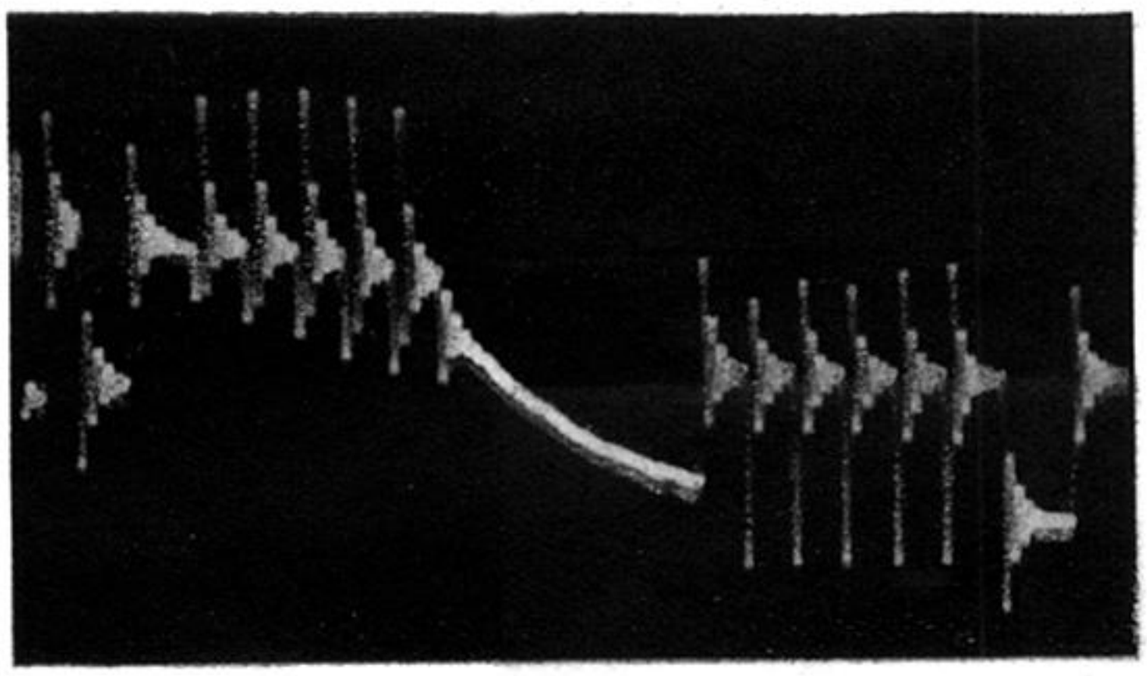
Simultaneous record of an experiment on the partially damped and fully damped galvanometers in circuit. The former, 3180, T., is shunted to $\frac{1}{4}$, oscillates 5 times per minute, and with the closure time of 7·5 seconds gives a swing-back = $\frac{2}{3}$rds the original swing. The latter, 3181, M., is not shunted, and has a falling-time of 12 seconds.

The two records, which exhibit the ordinary result of prolonged tetanisation, are given in explanation of the correction to be made for oscillation (3180, T.). In 3181, M., the closure-time being shorter than the falling-time, the negative variations do not reach their maximum. In 3180, T., the net after-effect is due to the state of nerve during the 2 to 3 seconds immediately after the $7\frac{1}{2}$ seconds excitation; in 3181, M., any positive after-effect would be due to the state of nerve during the last $\frac{2}{3}$rds of each minute. Such positive after-effect is better seen in plate 2177 (fig. 3B).

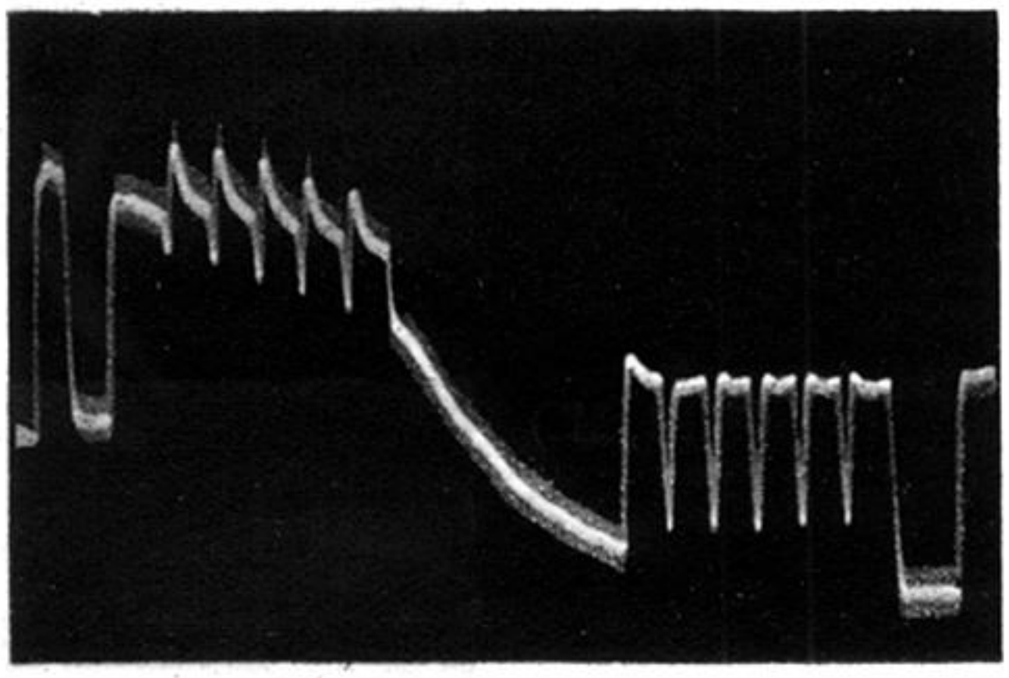
	Time.	I.	II.	(·6 I.)	II. net.	I./II.	0·001 volt.
	minutes.						
3180, T.	1	− 3·5	+5·7	(2·1)	+3·6	..	9·5
	2	− 5·3	+5·7	(3·2)	+2·5		
	3	− 6·0	+5·7	(3·6)	+2·1		
	5	− 6 0	+5·5	(3·6)	+1·9		
	12	− 6·5	+5·0	(3·9)	+1·1		
		Tetanisation, 10 units, 5 minutes $\alpha = -6{\cdot}5$ $\omega = +11{\cdot}5$					
	19	− 9·5	+5·5	(5·7)	−0·2		
	25	− 8·5	+5·0	(5·1)	−0·1		
	30	− 8·5	+5·0	(5·1)	−0·1		
	32	− 8·0	+5·0	(4·8)	−0·2	..	8·7
3181, M.	1	− 4	+4	..	..	..	13·5
	2	− 6	+2·5				
	3	− 6·5	+1·5				
	5	− 7	+1·2				
	12	− 7·5	+1				
		Tetanisation, 10 units, 5 minutes $\alpha = -10{\cdot}5$ $\omega = +17$					
	19	−11	0				
	25	−10	0				
	30	− 9·5	0				
	39	− 9	0	..	..	..	13·5

Fig. 70.

3188, T.



3189, M.



Simultaneous record of an experiment (influence of tetanisation on nerve in the second stage) on the partially damped and fully damped galvanometers in circuit.

	Time.	I.	II.	(·57 I.)	II. net.	I./II.	0·001 volt.
	minutes.						
3188, T.	0	− 3·2	+8·0	(1·8)	+6·2	..	15
	1	− 4·8	+9·4	(2·7)	+6·7		
	2	− 5·4	+9·6	(3·1)	+6·5		
	3	− 6·2	+9·5	(3·5)	+6·0		
	4	− 6·4	+9·4	(3·6)	+5·8		
		Tetanisation, 10 units, 5 minutes $\alpha = -6{\cdot}5$ $\omega = +13{\cdot}5$					
	11	−12	+4·0	(6·8)	−2 8		
	12	−12	+5·5	(6·8)	−1·3		
	13	−11·5	+5·3	(6·6)	−1·3		
	14	−11·5	+6·0	(6·6)	−0·6		
	15	−11·5	+6·5	(6·6)	−0·1	..	14·5
3189, M.	0	− 2	+4	..	..	..	14
	1	− 3·3	+3·5				
	2	− 3·8	+3·2				
	3	− 4·2	+2·8				
	4	− 4·5	+2·5				
		Tetanisation, 10 units, 5 minutes $\alpha = -4{\cdot}8$ $\omega = +11{\cdot}5$					
	11	− 9·3	0				
	12	− 9	0				
	13	− 9	0				
	14	− 8·8	0				
	15	− 8·5	0	..	..	..	13